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THE ROENTGENOLOGIC DIAGNOSIS OF PNEUMOCONIOSIS (SILICOSIS) AND USE OF THE "ELECTRIC EYE" TO DETERMINE REGIONAL DENSITIES[1]

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FOREWORD

SUCH a survey of this subject as we have been able to make indicates that cases diagnosed as silicosis differ in various communities and even in various industries in the same community. We believe, therefore, that a comprehensive approach to this problem should be based on a study of roentgenologic and pathologic findings observed in various industries and widely separated communities. To those who have aided us in the assembling of this material we wish to give special credit, and to express our sincere appreciation. The list is too long for us to include them as collaborators, but we do wish to mention at the very beginning that without the co-operation of the following men this investigation could not have been completed.

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Importance of the Roentgenologic Diagnosis of Pneumoconiosis (Silicosis).—It is almost universally conceded that the diagnosis of pneumoconiosis (silicosis) depends upon the accurate interpretation of satis-

[1] This is one of a series of papers contributed by friends and former pupils of I. Seth Hirsch, M.D.

factory roentgenograms, preferably stereoscopic ones. The British, in South Africa, early recognized this, and that America purpose to devise some method or standard of interpretation so that the clinician would not have to lean heavily on an ex-

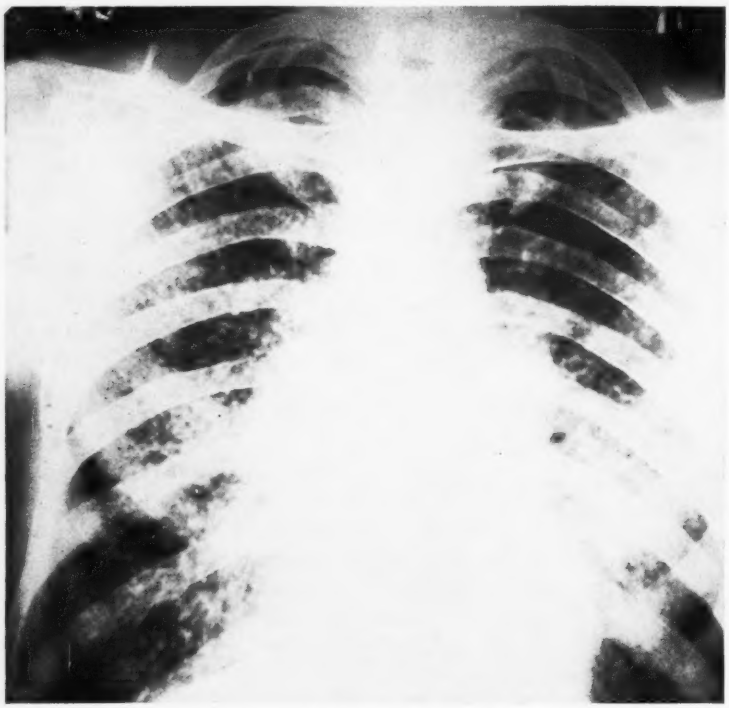


Fig. 1. Roentgenogram showing bilateral symmetrical mottling (nodulation). Classified by Dr. Smith, of South Africa, as "primary." This film is classical of some 200 roentgenograms diagnosed as silicosis by roentgenologists in various sections of this country and Canada, and now registered with the J. B. Pierce Foundation. (*Lent by Dr. McArthur, Noranda, Quebec.*)

The vast majority of these roentgenograms can be diagnosed as pneumoconiosis as rapidly as they can be placed upon the light box. Cases less extensive than this were classified by Dr. Smith as "ante-primary," and cases less extensive than the "ante-primary" ones were classified as "MFU++" or "MFU+." More extensive cases were classified as "secondary," and the terminal stage was classified as "infected," either tuberculous or non-tuberculous.

agreed with the British was manifested by a report made to the United States Government (1) by a committee of physicians, in which it was stated that "only the physician who has examined the subject, has obtained the occupational history of adequate exposure to silica dust, and has before him a suitable roentgenogram of the chest should make the diagnosis of silicosis."

In a later paragraph, the committee making the report stated that it was their perienced roentgenologist for the diagnosis of silicosis. But the diagnosis of pneumoconiosis (silicosis) is not unlike the detection of a criminal. In some cases the evidence is so simple that the criminal can be discovered by the merest tyro of a detective, while in other cases, only the most elaborate correlation of seemingly unimportant findings enables the G-men to apprehend the criminal. We believe that the elimination of an x-ray specialist in the diagnosis of pneumoconiosis (silicosis) has

led to unfairness both to the laborer and the industry in which he labors, and that a more elaborate effort to correlate roentgenologic and pathologic findings is imperative, if individuals and companies are to be treated fairly.

Roentgenograms of the lungs of subjects who have inhaled quantities of certain mineral dusts over long periods of time show findings that are not observed in roentgenograms of those not so exposed. This fact is clearly demonstrated by comparing roentgenograms of groups of subjects in dust-hazard industries with roentgenograms of groups of banking clerks.

The lungs of those exposed to certain mineral dusts show morbid changes in the form of deposits of tissue composed mostly of collagen, though they may also contain dust-laden phagocytes, desquamated cells from the lining membrane of the alveoli, disintegrated white blood cells, and débris within the alveoli. The amount of morbid tissue may be so slight that it is not discernible to the naked eye, and even so slight as to be overlooked upon microscopic examination. On the other hand, the amount may be so great as to practically fill the thoracic cage.

Dust-laden air is inhaled and the larger dust particles are expectorated because of the action of the cilia, but the smaller particles find their way to the alveoli and are mopped up by the phagocytes. The distribution of the morbid tissue, therefore, whether slight or extensive, is bilateral and relatively symmetrical. Much of it is laid down along anatomic structures, especially in the early stage of the process, but nodules, or spherical whorls of collagen, which are the most familiar type of morbid change, do not conform to anatomic structures.

Even slight deposits of morbid tissue, scattered discretely through both lungs, cause increased density of the lungs which is readily discernible on the roentgenogram (Figs. 1 and 18) when one's eye is trained to see it or the standard photometer or "electric eye" is employed to record it. Massive deposits of morbid tissue are so large and dense that their shadows on the roentgenogram may obscure the outline of the heart or even obscure the ribs. Morbid deposits in the lung occupy space that would otherwise be filled with air, and, therefore, cast shadows on the roentgenogram. Such shadows, referred to as "roentgenologic findings," correspond in size, shape, and distribution to deposits of morbid tissue in the lung.

Roentgenologic findings may be considered under two primary groups: (*A*) structural changes which cause a characteristic pattern on the roentgenogram; (*B*) regional distribution of densities on the roentgenogram, regardless of their pattern. We shall consider first the pattern approach.

A.—PATTERN APPROACH

Morbid tissue deposits in pneumoconiotic (silicotic) lungs fall into four main patterns, illustrated by the schematic drawings in Figures 2, 3, 4, and 5. Roentgenologic shadows resulting from and corresponding to these four patterns of morbid tissue deposits are illustrated in Figures 6, 7, 8, and 9. Although different dust irritants may be responsible for these four patterns, two or more of these findings are often seen in the same roentgenogram. Since they are more readily described and comprehended when considered separately, we shall take them up as follows:

1. Accentuated hilar and linear markings.
2. Nodules—small white spots on a dark background.
3. Pockmarks—small dark spots surrounded by light rings.
4. A general nondescript haze, or cloudiness.

1. *Accentuated Hilar and Linear Markings of Pneumoconiosis (Silicosis).*—The term "accentuated hilar and linear markings" is applied to a group of roentgenologic findings observed by Pancoast and described by him as constituting "the perivascular–peribronchial–lymph-node manifestation of pneumoconiosis."

The root or hilus of the lung is composed of a pulmonary artery and vein, the right

and left bronchus and its immediate branches, lymphatic glands and structures, and the stroma supporting these structures. In roentgenograms of normal lungs, these structures are fairly clear-cut, and usually the blood vessels and bronchi can be readily identified. They cast a definite shadow compared with the well-ventilated lung in the peripheral third.

For the sake of convenience in studying the accentuated linear and hilar markings of pneumoconiotic (silicotic) lungs, we have divided the lung into three regions: (1) the central or hilar region; (2) the middle third; (3) the outer or peripheral third.

Roentgenograms of subjects exposed to relatively small amounts of certain mineral dusts often manifest a definite increase in the density and size of the hilar shadow. Increased density in the central or hilar region is due to a deposit of morbid tissue composed of phagocytes laden with mineral dust, and collagen laid down in the stroma of the lymphatic glands and structures adjacent to them. Hilar shadows on the roentgenogram may be clear-cut, and, in such cases, are interpreted as enlarged, circumscribed glands. On the other hand, hilar shadows on the roentgenogram may extend out along the branches of the large blood vessels and bronchi. These extensions or prolonged shadows are caused by phagocytes laden with mineral dust which have been dammed back or caught in a traffic jam on their progress to the hilus (Fig. 10-*A*), or due to envelopes of collagen around the larger blood vessels and bronchi (Fig. 10-*B*), or to a proliferation of fixed cells in these regions. When shadows of these prolongations are well defined on the roentgenogram, they project out into the lung along the anatomic structures of the blood vessels and bronchi.

Some subjects, exposed to essentially the same type and amount of exposure as those manifesting increased hilar markings, develop accentuated linear markings in the middle third of the lung. Here the markings run parallel to the medium-sized bronchi and blood vessels. They are shadows of morbid tissue laid down in the peribronchial and perivascular structures. This morbid tissue is caused by dust-laden phagocytes and a proliferation of fixed cells in the interstitial structures.

Accentuated linear markings may also occur in the peripheral third of the lung, and here their roentgenologic appearance is fine and lacy, in comparison with accentuated markings in the middle third. Accentuated markings in the peripheral third are caused by small deposits of collagen along terminal blood vessels and bronchi. Increased linear markings in this region often cause a general mottling which in the past has not been differentiated from fine nodulation and pockmarking, but when films are studied with these fine linear markings in mind, they can be differentiated readily from the other three roentgenologic patterns of pneumoconiosis (silicosis).

The migrating phagocytes are an important factor in the morbid changes which cause increased hilar and linear markings. They may be loaded with silica (nonopaque refracting crystals), or they may be loaded with other foreign-body flecks (opaque, non-refracting crystals), but in either event they are a contributing cause to the accentuated hilar and linear markings.

2. *Nodules—Small White Spots on a Dark Background.*—Roentgenograms of subjects exposed to dust hazards often exhibit small white spots on a black background which are referred to as "nodules." These small white discs were early recognized by the investigators of pneumoconiosis in South Africa, and were named "nodules" by them. Nodules may be fine or coarse. They are dense, clear-cut shadows of morbid tissue deposited in the parenchyma of the lung. They are bilateral and relatively symmetrical and more marked in the mid-lung fields.

The morbid tissue which forms the nodule is spherical, with a well-defined circumference. It is composed of layers of collagen laid down upon one another in whorls, much like the layers of an onion. A microscopic section of such a nodule

reminds one of the whorls in a finger print (Fig. 11-*A*). Two, three, four, or even five of these individual nodules may be connected by strands of collagen so that they form a conglomerate nodule (Fig. 11-*B*). On the roentgenogram, the shadows of the individual nodules are very small. The conglomerate ones are larger, but are observed to be composed of two or more isolated nodules, connected by collagen fibrils. (Whether the small nodules increase in size and eventually become the large, discrete nodule, or whether a group of small nodules coalesce and form one of the large nodules is problematical and does not come within the scope of this communication, but will be considered in another article devoted to the development of collagen in silicosis.)

Nodules vary in size in different patients and even in the same patient. The variation in size observed in different patients may be influenced by the character of the dust irritant in different industries. Individual discs may be so small as to be scarcely discernible to the naked eye, or as large as a grain of wheat. The larger ones are referred to as "coarse nodulation" and the smaller ones as "fine nodulation." The larger discs are extremely brilliant because of the solidity of the nodule, and they cause a symmetrical mottling of both lung-fields. On the roentgenogram, these appear to be closer together and far more numerous than they are observed to be on a cut section, for on a cut section one sees only the nodules that appear in one plane, whereas on the roentgenogram the shadows of all the nodules are recorded and appear on the flat surface of the film. This accounts for a remark frequently made by the pathologist to the effect that there are not nearly so many nodules as are shown on the roentgenogram.

Heretofore, the nodule has been considered pathognomonic of silicosis, and is the finding on which the definition is based. However, nodules such as shown in Figure 11-*A* are observed to contain only a few foreign-body flecks and the majority of these are opaque, non-refracting flecks (therefore, not silica). In our opinion, the nodule is pathognomonic of pneumoconiosis, not silicosis.

3. *Pockmarks—Small Dark Spots Surrounded by a Light Ring.*—Roentgenograms of subjects exposed to certain mineral dusts may show small dark spots surrounded by white rings, somewhat irregular in shape. This finding, which we have termed "pockmarking," has, for some reason, been overlooked in the past or confused with the mottling due to nodulation or peribronchial accentuation in the peripheral regions of the lung. This finding reminds one of the full-blown stage of chickenpox or smallpox, and we have, therefore, designated it as the "pocking" sign or "pockmarking."

On the roentgenogram, numerous small areas of diminished density cause black spots about one-eighth inch in diameter, and these are surrounded by circular areas of increased density. They are the direct reverse of the nodule, being black, in contradistinction to the white discs or nodules. Pockmarks are bilateral and relatively uniform in distribution, though more marked in the peripheral third of the lung. They may be more advanced on one side than the other.

It is difficult to account for pockmarks by recognized morbid changes (Fig. 12), but they are best understood by an intensive study of the markings in roentgenograms of normal lungs. In a well-timed roentgenogram of a normal lung, one can see the fish-net appearance in the periphery created by terminal blood vessels as they form their anastomosing loops from arteries to veins. These multiple small loops form the septa between terminal lobules of the lung. Such lobules can be noted on the surface of a removed lung when it is gradually inflated, many deflated lobules popping up as air is forced into them. This network of anastomosing veins and arteries is the anatomic, architectural pattern on which the pockmarks develop (Fig. 14). With the deposition of fine fibrils of collagen in the interstitial structures in pneumoconiosis (silicosis), the

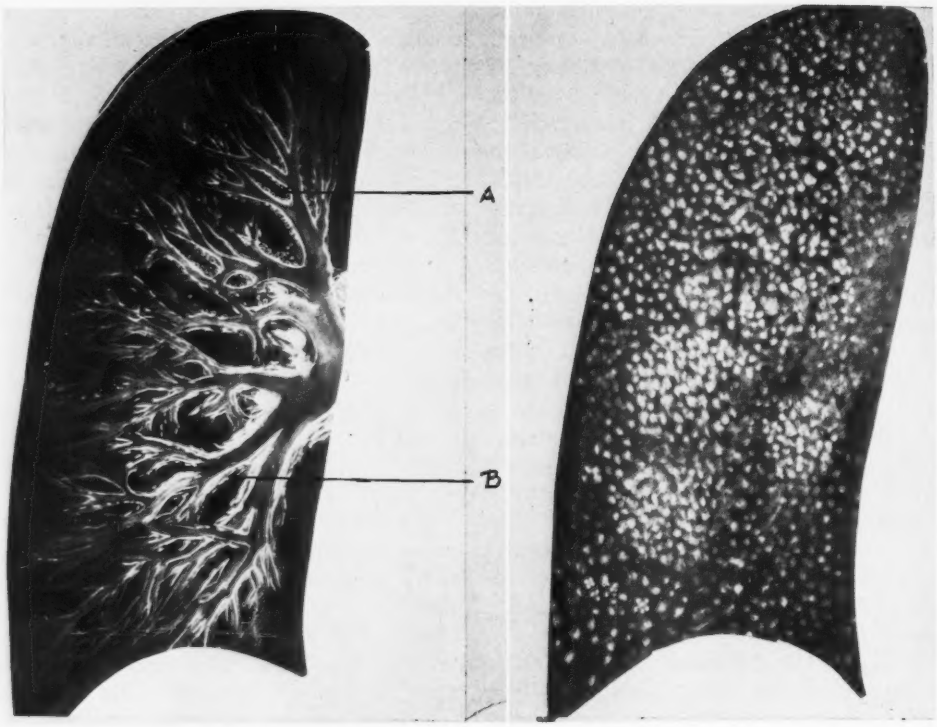


Fig. 2. Fig. 3.

Fig. 2. Perivascular–peribronchial–lymph-node manifestation of pneumoconiosis (Pancoast's disease). A schematic drawing presenting the author's conception of the combined roentgenologic and pathologic findings of this type of disease.

Accentuated hilar and linear markings are due to two pathologic findings—foreign-body flecks, and collagen deposits. Circles (*A*) indicate phagocytes which have clumped together foreign-body flecks that are opaque to both light and x-ray. Dashes (*B*) indicate collagen deposits laid down in the form of whorls or as laminæ around the blood vessels and bronchi. Both *A* and *B* accentuate the linear markings.

Although accentuated linear markings are a definite manifestation of pneumoconiosis, the lesion is of little or no clinical significance in early or even moderately advanced stages. The subject usually lives to a ripe old age without suspecting he has a lesion in the lungs.

Socially and economically, this type of lesion is a menace to both labor and industry. A patient who is perfectly able to work and wants to work may not be able to get a job because he has such a lesion. On the other hand, industry may be saddled with the compensation of a man with such a lesion who is perfectly able to work but does not want to. Legislatural acts fail to differentiate this type of pneumoconiosis which does not require compensation, from the more serious types of lesions which do deserve compensation at some stage in the life history of the disease.

Fig. 3. Nodular pneumoconiosis is the conventional type on which the diagnosis and even the definition of silicosis are based. Our observations indicate that these nodules, considered pathognomonic of silicosis, are not caused by silica. (Data substantiating this contention will be submitted in a subsequent article.)

Light and dark field examinations of these nodules reveal an overwhelming preponderance of black flecks that are not silica crystals, and only relatively few refractive crystals of silica, therefore, it would seem irrational to consider the few silica crystals as the etiologic factor in the development of these nodules. Nodules are composed of collagen, laid down like the layers of an onion; they are bilateral, symmetrical, and separated from one another by ventilated lung. Later they may grow together in round masses in the mid-lung field, resembling a pawnbroker's sign. Roentgenologically, they appear as white spots on a black background. The lungs may be literally shot full of these nodules without the patient having dyspnea or any other symptoms, and these patients, too, may live to a ripe old age, without knowing that they have nodular pneumoconiosis.

Socially and economically, such subjects are able to carry on hard labor at the prevailing wage. Should they be prevented from getting such jobs? Or lose the ones they have? Legislative acts are built primarily around this type of lesion, which has been considered silicosis.

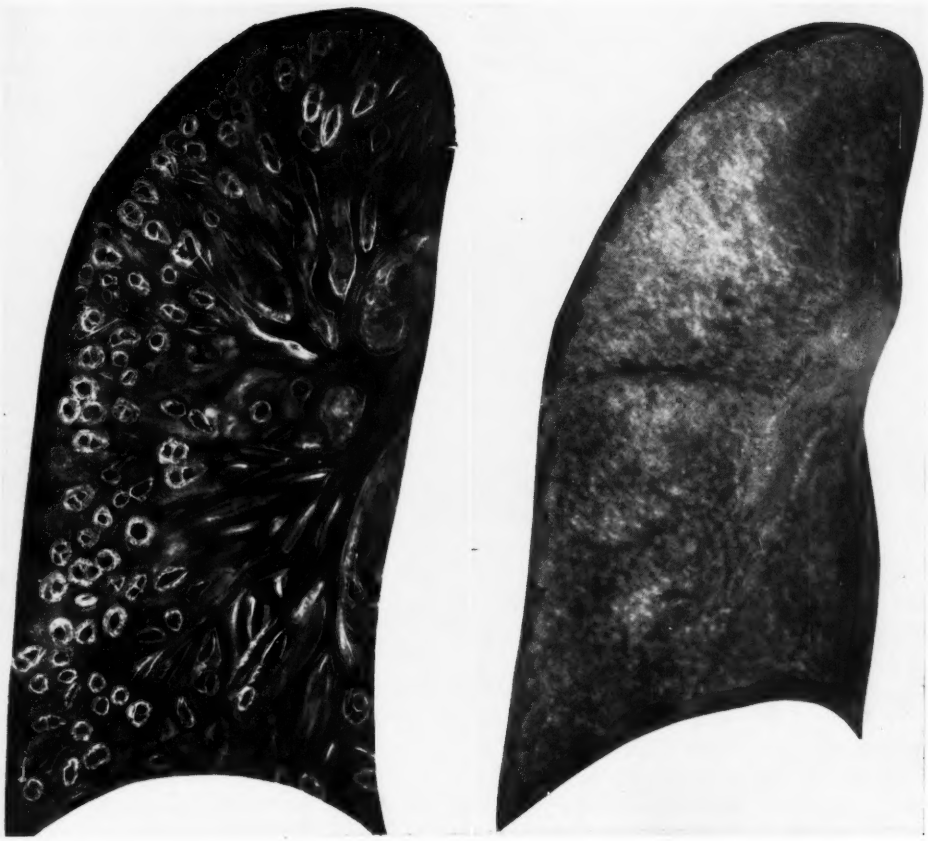


Fig. 4. Fig. 5.

Fig. 4. Pockmarking is the roentgenologic manifestation of a pathologic finding that has not been hitherto recognized, or which, at least, has not been differentiated from the other three types of pneumoconiosis.

Roentgenologically, pockmarking is evidenced by small black spots on a white background or surrounded by white rings. These black spots are caused by air cysts in the lung which are surrounded by lung rendered relatively dense from collagen laid down in the form of whorls or strands. The air cysts correspond anatomically with terminal lobes that are choked off from the bronchioles by the ball-valve action of the collagen laid down around the smaller bronchi.

In a microscopic section in which these air cysts are most marked, the opaque, non-refractive foreign-body flecks overwhelmingly predominate the transparent or translucent refractive silica crystals.

Air cysts are of more clinical significance than the nodules and are more likely to be associated with dyspnea and other symptoms. They should not, however, be used as the only criterion to determine whether or not the subject is incapacitated for work. Since these air cysts and the pockmarking which they cause have not been recognized before, they have played no part in legislatural acts. We believe, however, that in the future the presence of these air cysts in relatively dense lungs may play an important part in determining when compensation should begin.

Fig. 5. Rapidly developing or acute silicosis is evidenced roentgenologically by a general haze or diffuse cloudiness which obscures the normal markings of the lungs.

The perivascular and peribronchial deposits of dust-laden phagocytes and laminæ of collagen, and the nodular whorls and areas of massive collagenization are absent or extremely scanty in this type of disease, even in the terminal stage.

The general haze and diffuse cloudiness observed in the roentgenograms are caused by an incomplete filling or consolidation of the alveoli and air passages with phagocytes, and hypertrophied and proliferated cells, and also by a thickening of the walls of the alveoli themselves.

Economically and socially, these subjects present a most serious problem. They deserve adequate compensation as soon as the diagnosis is definitely established, or certainly as soon as dyspnea develops, which is relatively early. Injustices have been done in the past because subjects having this type of lesion were unfortunate in that they did not have the well-developed nodulation on which the diagnosis and definition of the disease are based. Since roentgenograms showed no nodulation, there was a tendency to consider that such cases were not silicosis and, therefore, were not "within the law."

normal network described becomes more coarse. It is the shadow of this increased collagen, laid like an envelope around terminal blood vessels and bronchi (Fig. 15), which causes the pockmarking on the roentgenogram (Fig. 8). When the pockmarks are superimposed over the drawing of the anatomic structures, the pathogenesis of the pockmark becomes startlingly evident (Fig. 4).

Pockmarks are sometimes observed also in the central or hilar portion of the lung, and there they are definitely established to be bronchi viewed on end, surrounded by a laminated layer or envelope of collagen.

As pneumoconiosis (silicosis) progresses, pockmarks may be obscured, just as the individual whorls or nodules are obscured by conglomerate nodules or massive collagenization. The mottling caused by the pockmarks is increased in density by a laying down of collagen until the center of the pockmark is not evident and the mass becomes a relatively solid area.

4. *General Haze or Cloudiness.*—Acute silicosis is manifested roentgenologically by a general haze or cloudiness which tends to obscure the normal markings. The findings are difficult to describe; there is a general cloudiness of the entire lung-fields. In localized areas, this general haze has developed into definite clouds, so that the appearance is not unlike a view from an airplane when we are looking down on white clouds and through the breaks in the clouds we see darker, less misty areas. The ventilation of the lung is materially diminished, and yet there seems to be no characteristic pattern observed on the roentgenogram. The accentuated hilar and linear markings, the nodules, and pockmarks are absent, or obscured by the lesion which causes the haze.

The general haze is caused by a variety of morbid changes (Figs. 13-*A* and 13-*B*): (1) thickening of the walls of the alveoli; (2) deposits of various types of material within the alveoli; (3) envelopes of collagen surrounding the smaller blood vessels and bronchi. Viewed microscopically, one observes envelopes of collagen around the larger and medium-sized blood vessels, and a few single or conglomerate whorls scattered through the lung, but these latter findings are usually slight and are obscured by the general cloudiness characteristic of the disease.

The first cause contributing to the general haze is the thickening of the walls of the alveoli. These may be thickened to eight or ten times their normal size, and their abnormal thickness is due to a laying down of collagen in certain localized regions, and an immense dilatation and engorgement of the capillary network forming the alveolar walls in other regions. In the regions in which the alveolar walls are thickened by collagen, the collagen has so constricted the capillaries that red blood cells are absent, and microscopic field after field may be studied without observing a single one. These are avascular areas. In other regions of the lung the alveolar walls are thickened to an equal degree by an immense dilatation and engorgement of the capillary network which forms them. These are hypervascular areas. There is a minimal amount of collagen in the walls of the alveoli, but there may be a seepage of the blood cells into the alveoli. The walls of the alveoli, thickened in these two manners, encroach upon the alveoli and diminish the ventilation of the lung, thereby contributing to the general haze.

A second contributing cause is the deposit of various types of material within the alveoli. Phagocytes, either empty or silica-laden, are deposited in large numbers in the alveoli. Cells of the membrane lining the alveolar walls are hypertrophied, proliferated, and may be desquamated into an alveolus. The alveoli may also contain an amorphous material in combination with the "cholesterol crystal clefts" described by Gardner (6).

A third cause contributing to the general haze is an envelope of collagen encircling the smaller blood vessels and bronchi.

All of these factors tend to diminish the ventilation of the lung and increase its solidity, but the lung still retains an amount

of ventilation, so that the solidity gives only a general haze rather than a dense shadow such as that caused by consolidation or interstitial collagenization of the nodular type.[2]

The general haze is bilateral and relatively uniform in its distribution. Poorly defined areas of increased density of the lung vary in size and are evenly distributed throughout both lungs. The absence of accentuated hilar and linear markings, of nodules, and of pockmarks often cause the roentgenologist to overlook the general haze, unless he is silica-minded, and even then he is apt to disregard the finding unless he is familiar with the morbid changes of acute silicosis. The general haze is a finding rarely observed in any other lesion. We believe that the roentgenologic problem of acute silicosis is its recognition in unsuspected cases, rather than its differentiation after the lesion is once recognized.

The reader may note that in discussing the manifestations above we have not hesitated to use the term "silicosis." This is because microscopic sections of such lungs show an overwhelming amount of silica (non-opaque refracting crystals) and relatively few foreign-body flecks of other elements. An analysis of lungs showing roentgenologic nodulation revealed that they contained an overwhelming preponderance of opaque non-refracting crystals, and when referring to them we use the term "pneumoconiosis (silicosis)."

Massive Collagenization.—Thus far we have analyzed four roentgenologic findings with their corresponding pathologic findings. We have discussed and illustrated the four patterns in cases of pneumoconiosis (silicosis) in which the disease had progressed sufficiently for the pattern to be well marked but had not developed to such a degree that the pattern was obscured on the roentgenogram. Often three or even four of the roentgenologic manifestations described may be observed on the same roentgenogram, but one pattern usually predominates so that the case is readily classified.

As pneumoconiosis (silicosis) progresses, the morbid tissue is increased by massive deposits of collagen. These cast dense roentgenologic shadows which obscure the nodulation and pockmarking observed in earlier stages of the disease. The collagen masses may be made up of large numbers of nodules closely packed together (Fig. 7), or of strands of collagen so closely packed as to obliterate the pattern (Fig. 19).

Massive collagenization may develop in three regions of the lung:

(1) Mid-lung Field. A "pawnbroker's sign" may be formed by three clumps or masses of collagen deposited in the right mid-lung field where the lobes of the lung join each other (Figs. 7 and 8). They are arranged one above and two below, and thus give the appearance of a pawnbroker's sign. Perhaps the anatomic structures formed by the three lobes of the lung account for these dense masses. When dense masses of collagen form on the left side, one of the masses or balls of the "pawnbroker's sign" is usually absent.

(2) Pleura. Massive deposits of collagen may develop in the pleura at the *apex of the lower lobe* (Fig. 17). There they form a mass with a clear-cut upper surface that arches outward and downward from a region near the spine, opposite the head of the fourth rib. Seen on the roentgenogram, the upper surface of this shadow is well defined and reminds one of the dome of the diaphragm. The under surface of this arch is irregular and fades into the mottling of the parenchyma of the lung. (The mottling, of course, is caused by a diffuse deposit of collagen in the parenchyma.)

(3) Upper Lung-fields. Masses of collagen having no characteristic pattern may develop in the upper lung-fields. They vary in size from one to two centimeters, in instances in which only a few nodules have formed into a clump too large to be considered a conglomerate nodule, up to

[2] A more detailed description of the morbid changes which cause the general haze of acute silicosis are described in an article entitled "Dyspnea of Silicosis: What Causes It?" which will be published in an early issue of the *Journal of the American Medical Association*.

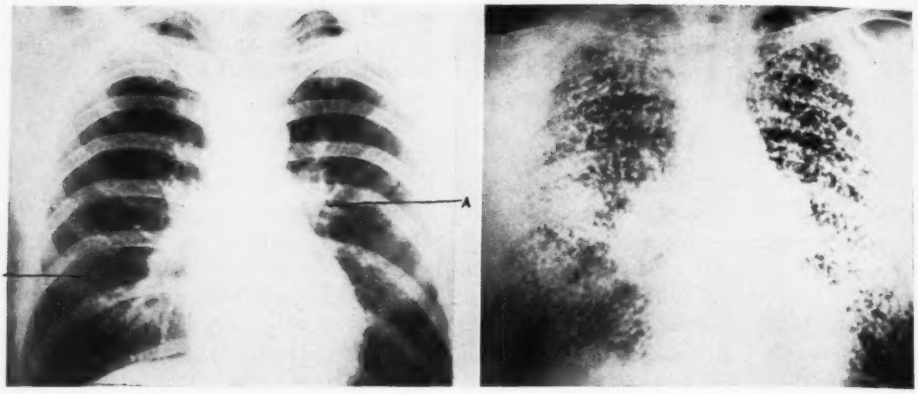


Fig. 6. Fig. 7.

Fig. 6. Roentgenogram showing enlarged hilar glands, *A*, and accentuated linear markings, *B*. This case was classified by Dr. Smith, of South Africa, as follows: "MFU. This is found in 30 per cent of the cases not underground." The author of this article has found the condition existing in buffers and grinders and employees of certain mining industries. It is shown especially in 17 roentgenograms contributed by Dr. Bridgers, of Pennsylvania, and Dr. Derr, of Maryland, to the silicosis registry described under Figure 1. (*Lent by Dr. McArthur, Noranda, Quebec.*)

Fig. 7. Roentgenogram showing small white spots on a black background (nodulation). Nodules are disseminated throughout both lungs, and are clumped into a "pawnbroker's sign" in the right lung-field. This patient had no dyspnea and no symptoms. The lesion was found accidentally while the patient was being examined for a fractured rib. This case of nodulation without symptoms is not an exception but is the rule. We have 53 cases with nodulation and practically all of them are free from dyspnea and other symptoms. (*Lent by Dr. Klein, Perth Amboy, New Jersey.*)

such a size as to fill the upper portion of the thoracic cage. They are almost always bilateral, but they may be more extensive on one side than the other.

Ingrafting of Infection—Pyogenic or Tuberculous.—As areas of massive collagenization increase, the blood supply is impaired (Fig. 20). Gross inflammatory lesions may develop in the morbid tissue that hitherto has not been inflammatory. Gross infection—pyogenic, tuberculous, or both—may be ingrafted on the areas of massive collagenization, or develop in the areas of the lung rendered avascular by small deposits of collagen that constricted the capillaries, as in the case of acute silicosis.

Pneumoconiotic (silicotic) collagen does not contract, but the collagen of tuberculosis and other inflammatory lesions does. Massive collagenization before infection sets in does not contract like reparative, fibrous connective tissue—the trachea is not retracted to one side, the interlobar fissure between the middle and upper lobes is not displaced, and the normal markings of the lung, so far as they can be recognized, are not altered in position. Tenting of the diaphragm and obliteration of the costophrenic angle (Fig. 22-*A*), and obscuring of the left border of the heart are often observed when an inflammatory process is ingrafted upon the massive collagenization of pneumoconiosis (silicosis). (The outline of the heart may be obscured merely by massive collagenization or a number of nodules.)

Whether tuberculous or non-tuberculous infection predominates in these late stages of pneumoconiosis (silicosis), and the question of when the process changes from one of simple massive collagenization to that complicated by infection are controversial problems which will be considered in a special article devoted to those subjects. Inflammatory and tuberculous processes do not develop in pneumoconiosis (silicosis) until massive collagenization has occurred. The contention that tuberculous infection develops in the earlier stages, when patterns of accentuated hilar and linear markings, nodules, or pockmarks are seen on the roentgenogram, was not con-

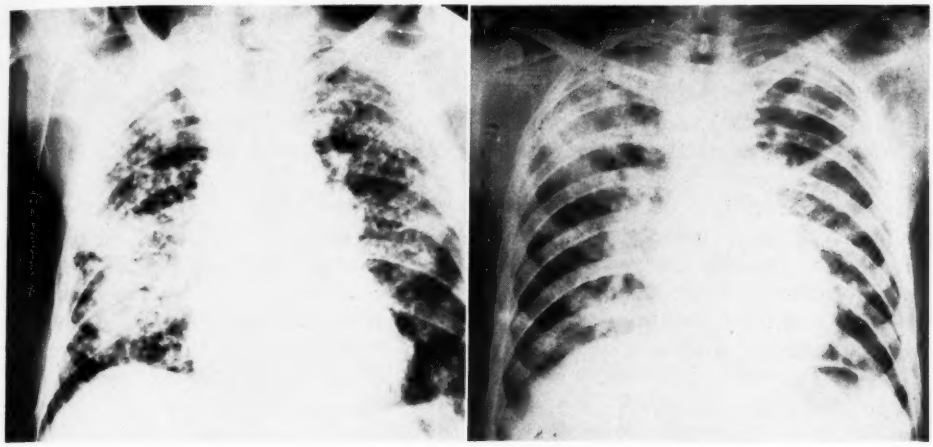


Fig. 8. Fig. 9.

Fig. 8. Roentgenogram showing small black spots surrounded by white rings (pockmarking). These remind one of chickenpox or smallpox. The small white areas of massive consolidation also show small black spots within their confines. This patient, with no more involvement than that shown in Figure 7, had marked dyspnea. (*Lent by Dr. W. G. Cole, New York City.*)

Fig. 9. Roentgenogram shows a general haze and diffuse cloudiness obscuring the normal lung markings. The general haze is far less dense than the collagen, observed in Figure 8, and the nodulation, shown in Figure 7, is singularly absent. There is no pockmarking.

This patient with acute silicosis had extreme dyspnea at the time this examination was made, in spite of the fact that there was no nodulation, and he died 20 months after the initial exposure. (*Lent by Dr. Harless, Gauley Bridge, West Virginia.*)

firmed by our study of films from various regions of the country and various dust-hazard industries.

Cavities and Localized Pneumothoraces.—Cavities in pneumoconiosis (silicosis) may be formed by a breaking down of the tuberculous or pyogenic infection or a combination of the two, and they occur in the upper third of the lung (Fig. 21). In our opinion, the process which precedes the cavity is either the breaking down of a tuberculous lesion in which mixed infection has occurred or the breaking down of a non-tuberculous inflammatory lesion.

In pneumoconiosis (silicosis) there are three types of cavities which may occur in the parenchyma: (1) a cavity caused by a breaking down of a tuberculous or non-tuberculous area of collagenization; (2) a spontaneous, localized pneumothorax usually near the apex; (3) split pleura (Fig. 22-*b*). This is a roentgenologic finding characteristic of pneumoconiosis (silicosis), which we have seen high up on the lateral wall about two or three inches from the apex of the lung. The visceral pleura is markedly thickened by gobs or strands of collagen laid down as laminæ. The outer portion is firm because of its previous anatomic structures, and the part adjacent to the lung is firm because of the normal lung structures it invades, but the central portion is composed of gobs or strands of collagen which are friable. Therefore, the inner portion of the thickened pleura breaks away from the outer portion, and air fills this cavity, causing the "split pleura" observed on the roentgenogram. This finding resembles either a localized pneumothorax or a cavity very close to the chest wall.

DIFFERENTIAL DIAGNOSIS

The roentgenologic findings of pneumoconiotic (silicotic) lesions must be differentiated from those of numerous pulmonary lesions. We shall discuss these in the same order observed in a discussion of the four roentgenologic patterns.

1. *Differential Diagnosis between Accentuated Hilar and Linear Markings of*

Pneumoconiosis (Silicosis) and Other Lesions.—Accentuated hilar and linear markings occur in many pulmonary lesions, and the problem of differentiation is a difficult one. However, when roentgenologic findings are combined with clinical evidence the differentiations may be made with relative accuracy. (As we shall see later, the differentiation is not important from a clinical or compensation standpoint.)

(*a*) Pneumonia. Increased linear markings due to pneumonia are usually unilateral or more marked on one side than the other. One may have an extension of the peribronchial markings as a residual lesion after a bronchopneumonic lesion, but these are usually unilateral, and diminish from time to time in intensity. A clinical history of nasal infection is obtainable in the case of pneumonia.

(*b*) Post-nasal Drippings. These may cause increased peribronchial markings, but they are usually limited to both lower lobes, that is, to the descending bronchi on both sides.

(*c*) Chronic Passive Congestion from Cardiac Decompensation. The increased linear markings of this lesion closely resemble those of pneumoconiosis (silicosis), being bilateral, but in such cases there is a definite history of a cardiac lesion, and the roentgenograms usually reveal an enlarged heart.

(*d*) Neoplastic Lesions. Metastases, lymphatic neoplasms, and Hodgkin's disease all present increased hilar shadows almost identical with those due to pneumoconiosis (silicosis). They may be bilateral and extend out along the branches of the larger blood vessels as do those of the pneumoconiotic lesion, but the clinical histories, the presence of enlarged glands in other parts of the body, or the x-ray therapeutic test enables one to differentiate them.

(*e*) Bronchiectasis. This may present bilateral or unilateral accentuated linear markings, but the accentuation is more marked in both lower lobes. Differentiation is readily made by clinical history—bronchiectasis being accompanied by cough with profuse expectoration. A definite roentgenologic differentiation may be made by the injection of iodized oil.

2. *Differential Diagnosis between Nodules of Pneumoconiosis (Silicosis) and Nodules Due to Other Lesions.*—(*a*) Pulmonary Tuberculosis. Miliary tubercles differ from nodules in their individual appearance and in their distribution throughout the lung. While the individual tubercles may be identified and counted, they have a hazy, poorly defined circumference because of the exudative tissue of which they are composed, in contrast to the clear-cut and well-defined nodules of pneumoconiosis (silicosis). The distribution in the case of pulmonary tuberculosis is usually unilateral with a cross-infection on the opposite side. The primary lesion of tuberculosis is usually a fan-shaped or cone-shaped involvement at the apex, and though one may find a cross-infection in the mid-lung field of the opposite side, the lesion is more extensive on one side than the other. A clinical history of temperature and cough in tuberculosis is a further aid in differentiation, for in the early stages of pneumoconiosis (silicosis) the patient is singularly free from symptoms.

(*b*) General Acute Miliary Tuberculosis. A clinical history combined with a series of films, made three or four days apart, will readily differentiate these two lesions, for in acute miliary tuberculosis the disease progresses rapidly and the clinical evidence is definite.

(*c*) Fungi of Yeast Infections. Roentgenograms of yeast infections resemble those manifesting pneumoconiotic (silicotic) nodules, but the yeast growths are not as dense to the x-ray as the pneumoconiotic nodules. One must often depend on the clinical history, sputum examination, and other factors for a differential diagnosis. The following quotations from a comprehensive article by Pancoast and Pendergrass (5) may aid in differential diagnosis.

"Loptothrix Infection. We have encountered one case of this kind presenting an appearance quite similar to nodular silicosis, but the diagnosis was readily made by culture from the sputum, and there was no occupational history.

"Sporotrichosis. We have examined one case of this infection in which the appearance was so identical with that of the nodular phase of silicosis that we could not be convinced that the condition was not the latter except by the lack of exposure and by subsequent observations at autopsy. Miliary tuberculosis was also considered as a possibility and, in fact, the alternative."

We would add that although yeast infections are often bilateral, and frequently symmetrically bilateral, their shadows have a tendency to follow the pattern of the linear markings of the lung and that these markings are more evident at the hilus and base than in the mid-lung fields.

(*d*) Neoplastic Metastases. The shadows of certain diffuse metastases, especially carcinoma metastases, resemble the shadows of nodules of pneumoconiosis (silicosis) especially when the latter lesion has developed to the point at which there are patchy areas of collagenization. Without a clinical history or knowledge of the primary lesion, the two are most difficult to differentiate. The metastases may be unilateral or bilateral and distributed with relative uniformity. A series of films will reveal the rapid development of the metastatic lesion in comparison with the slow progression of the pneumoconiotic (silicotic) lesion.

The metastases of certain types of sarcoma resemble the embryonic yolks of chickens' eggs. They are clear-cut, round, or oval, much more dense than patchy areas of nodular collagenization, and they differ in their distribution, which is less symmetrical and more inclined to involve the lower lobes. The metastases vary markedly in size. Many of them are centrally located and appear as extensions of the hilar shadow.

(*e*) "Actinomycosis [5] may occasionally present an appearance like that of nodular silicosis, although the nodules are usually larger and less numerous in comparison with it and similar infections."

(*f*) Lobar Pneumonia. This differentiation is readily made because pneumonia rarely presents a bilateral, or, at least, a symmetrically bilateral involvement. Moreover, the clinical history in the case of pneumonia is definite—temperature, cough, expectoration, etc., while in pneumoconiosis (silicosis) the patient is free of symptoms until advanced stages or until some other type of lesion is ingrafted upon the pneumoconiotic one.

3. *Differential Diagnosis between Pockmarks of Pneumoconiosis (Silicosis) and Pockmarks of Other Lesions.*—One must depend upon location and distribution to differentiate the pockmarks of pneumoconiosis (silicosis) from those due to other lesions.

(*a*) Lobular Pneumonia. Pockmarks of pneumonia are readily differentiated from those of pneumoconiosis (silicosis), because the former are unilateral and in the central portion of the lung, whereas the latter are bilateral and found chiefly in the peripheral third of the lung.

4. *Differential Diagnosis between the General Haze of Acute Silicosis and the General Haze of Other Lesions.*—The symmetrical, bilateral general haze is a finding rarely observed in other lesions. It is a nondescript finding which may be confused with some types of unresolved pneumonia, but as we stated before, when discussing the pattern, the absence of accentuated hilar and linear markings, nodules, or pockmarks makes the roentgenologic problem the recognition of silicosis in unsuspected cases, rather than its differentiation from other lesions.

B.—REGIONAL APPROACH

The first approach to the roentgenologic diagnosis was based on the pattern shown on the roentgenogram. The second approach is based on the regional distribution of the various morbid tissues, regardless of their pattern. Regional distributions are best observed by viewing the roentgenogram at a distance, squinting the eyes so that one does not observe the pattern but considers only the densities of various portions of the lung compared with each other. For the study of these regional distributions each lung is divided as follows: (1) the apex—the region just below

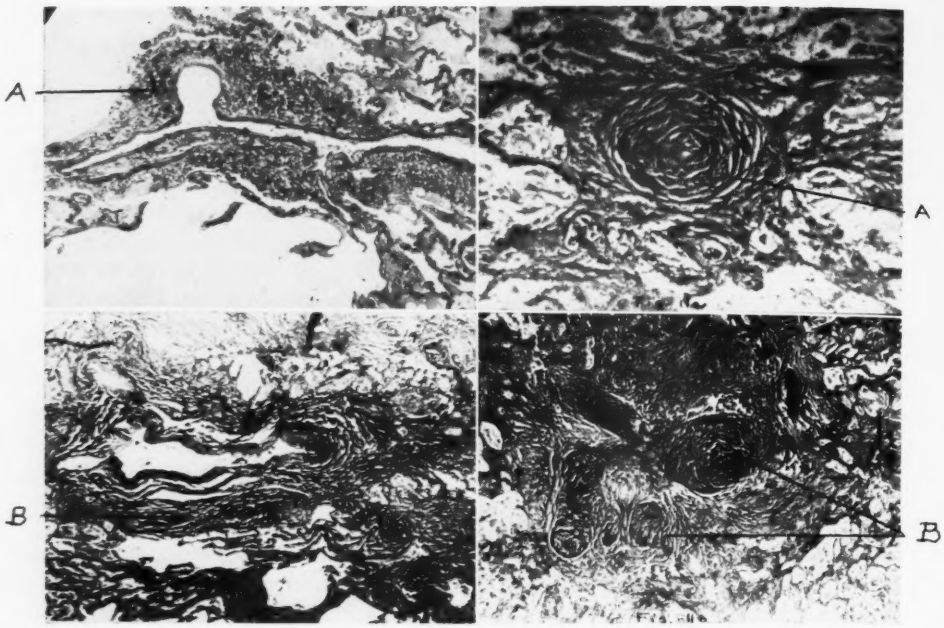


Fig. 10. Fig. 11.

Fig. 10. Shows the two types of morbid tissue which cause an accentuation of the peribronchial and perivascular structures. *A* represents huge quantities of phagocytes laden with black dust and deposited in the lymphatic structures parallel with the blood vessel which is cut longitudinally. Note the increased mass that forms almost a nodule where the blood vessel divides. This type of pathology was represented by circles in Figure 2. *B* represents laminæ of collagen laid down alongside a blood vessel which is cut tangentially. The collagen has a definite laminated appearance, as if the blood vessel were wrapped in cellophane. The thickness of the wall of the blood vessel is evidenced by the dense band adjacent to its lumen. This type of pathology was represented by dashes in Figure 2.

Fig. 11. Shows isolated and conglomerate nodules composed of whorls of collagen laid down like the layers of an onion. The nodules may be single (*A*), or several nodules may be clumped together to form a conglomerate nodule (*B*). Strands of collagen are observed between the whorls in the conglomerate nodule.

the clavicle; (2) the mid-lung field—between the third and sixth ribs anteriorly; (3) the base—extending from the mid-lung field to the diaphragm.

USE OF THE PHOTOMETER OR "ELECTRIC EYE"

Events Leading to the Use of the Photometer.—A chronological story of our experience in using the photometer or the "electric eye" for detecting regional densities in roentgenograms of pneumoconiosis (silicosis) may be of special interest (Fig. 23). In 1936, when we first began our study of the silicosis problem, we consulted the roentgenologic authorities in this country, namely, Pancoast and his associate, Pendergrass. Unfortunately, Pancoast's health prevented us from discussing the subject with him, but Pendergrass showed us the mass of roentgenologic material which he and Pancoast had used in their investigations and the preparation of their articles. Most of their better roentgenograms had been assembled for teaching purposes, and had been used as illustrations in their published articles, but we were delighted with an opportunity to observe the original films, which were, of course, much more satisfactory than the half-tone reproductions.

After observing these with Pendergrass, we asked what types of morbid changes caused these findings, and he drew a sketch of the whorl which he referred to as "the fibrosed nodule." He continually referred

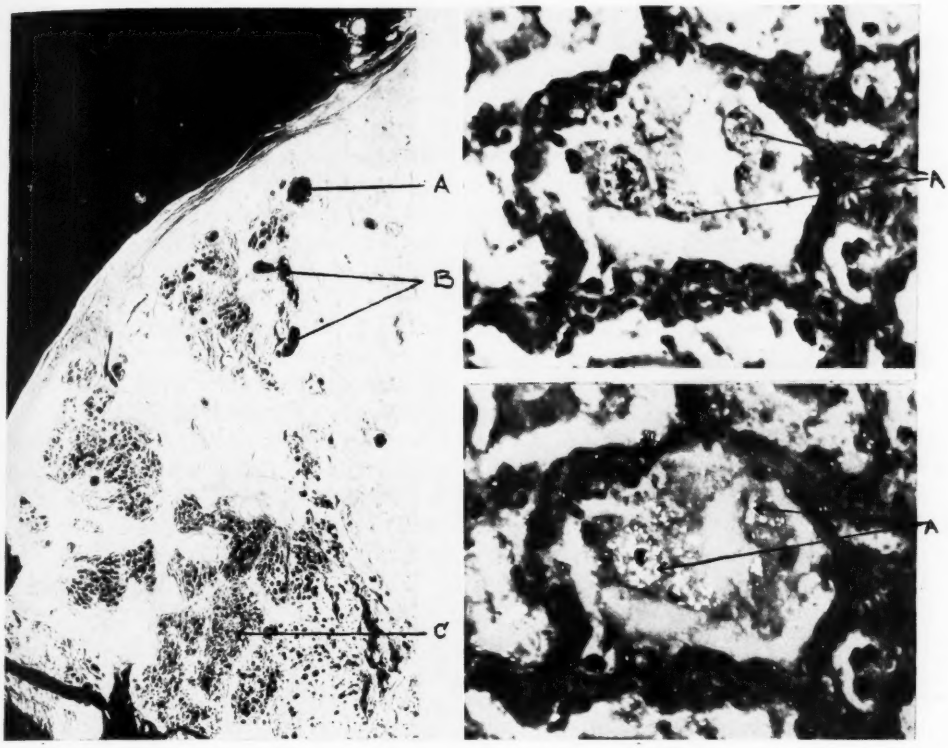


Fig. 12 (*left*). This is a negative print, *i.e.*, a direct enlargement on paper from the original microscopic section. Therefore, the dense areas in the slide appear as white areas in the print to correspond with the roentgenologic findings. This shows the air cysts composed of dilated terminal lung lobules. The exit of the air from these lobules is obstructed by the ball-valve action of collagen around the bronchiole (*A*). Air passages or bronchioles may likewise be dilated (*B*). *C* represents the alveoli in a relatively uninvolved region.

Fig. 13-*A* (*upper right*). An alveolus with a markedly thickened wall containing phagocytes which latter have mopped up opaque foreign-body flecks (*A*).

Fig. 13-*B* (*lower right*). Same alveolus viewed with dark field with polarized light shows huge quantities of refracting crystals (silica) which appear as white spots (*A*) within the phagocytes. *Silica is not observed in the light field* (*Fig. 13-A*) *but is very prominent in the dark field* (*Fig. 13-B*).

to Gardner, of Saranac Lake, as the pathologic authority, and spoke also of Dr. Adelaide Smith, of the Industrial Hygiene Department, New York City, suggesting that we contact her, which we later did with profit.

We then began to obtain roentgenograms. From a casual study of fifty or sixty of the peribronchial and perivascular type viewed individually, we were unable to recognize roentgenologic evidence of the disease, but when thirty or forty of these were set up on light boxes at the same time, it was apparent that in these films the mid-lung fields adjacent to the hilus showed increased density, and that these densities, although well marked, were not as clearly circumscribed as the densities of tuberculosis, either in the stage of consolidation or infiltration.

For the purposes of further study, we began to make lantern slides. In this connection we used the Weston Photometer, composed of a milliammeter and a selenium cell, which when exposed to the light allows the electric current to record a higher registration on the meter. This accurate instrument is used to determine the amount of light that strikes the object the operator is photographing. It may also be used to

determine the amount of light that passes through a negative, so that one may know the length of exposure to be used when making lantern slides or reductions of x-ray films.

In using the photometer for this purpose, it soon became evident that different portions of the chest films which we were reproducing, or of which we were making lantern slides, showed great variations as recorded on the meter. When the photometer was placed where the transmitted light passed through the heart, there was an extremely high reading, somewhere around 600 or 700. As we placed the photometer over various portions of the lung-fields, we were aware of a far greater variation in density than was apparent when one casually looked at the film by transmitted light.

We compared the photometric readings in various portions of pneumoconiotic (silicotic) roentgenograms with readings of normal lungs, tuberculous lungs, and lungs showing other pathologic lesions, and found that they varied from all of these. A specific recording of the findings in the fifty or sixty cases referred to was made. The recorded figures scarcely attracted attention, but when we began to place these figures on a chart and arrange the readings, it became apparent that with a certain arrangement we could obtain a spectacular and illuminating graphing.

Graphing of the Photometric Readings.—The readings of a standard Weston Photometer run from 0 to 1,000. An illuminating box was used and a bulb secured which gave a reading of about 900 without any film interposed, or 600 or 700 through the heart. In the lung-fields where there is an appreciable amount of air or ventilation, the normal structures of the lung obstruct x-rays to a lesser degree than does the heart, and, therefore, the lung-fields appear less dense on the roentgenogram than in the region of the heart. If, as described before, the lung is divided into apex, mid-lung fields, and base, the photometer reading in the mid-lung fields would be higher than at the base or apex. As the heart obstructs more rays than does any other portion, the photometer recording is highest here.

In the normal lung, the base is well ventilated and shows a low reading on the photometer. The mid-lung fields show approximately the same resistance to penetration as the base, being a trifle more or a trifle less dense. At the apex, just below the clavicle, where the lung is smaller and not so thick and the muscles heavier, the density of the plate is greater. To make the reading complete, the photometer reading is made over the mid-line in the region of the trachea. Typical photometric readings of a normal lung would read somewhat as follows: heart—500; base—100; mid-lung field—125; apex—135; trachea —450. The relative variations here show that the ventilation in each region is normal (Fig. 24).

We have simplified this reading by putting a rheostat in circuit, so that the transmitted light can be altered and the light through the heart recorded on the meter as 500, regardless of over- or under-exposure of the roentgenogram. Having one constant factor, the findings are more readily charted and compared.

By charting these figures in the manner illustrated, we obtain a chart somewhat resembling a capital *V*. This is the characteristic chart of the normal lung, with which chart pneumoconiotic (silicotic) lungs should be compared.

Characteristic Graphs of Peribronchial Pneumoconiosis (Silicosis).—Peribronchial pneumoconiosis (silicosis) which is manifested by increased linear markings, bilateral and relatively symmetrical, gives photometric readings which, when graphed, become of great value in showing the regional variations in lung densities in the middle, upper, and lower fields. Throughout this article we have referred to roentgenologic findings in terms of lung densities rather than photographic densities, but we are now dealing with measurements of photographic densities on the roentgenogram, and it must be borne in mind that the areas of greatest lung density are evidenced by diminished density of the

roentgenogram, and, therefore, give higher readings on the photometer.

In typical cases of peribronchial pneumoconiosis (silicosis) with accentuated linear markings, we have readings somewhat as follows: heart—500; right base—40; right lung-field—185; right apex—105; trachea—425; left base—40; left lung-field—200; left apex—100.

If these are placed on a chart, with the regions recorded from left to right and the numbers of the photometer recorded in the vertical column, we obtain the characteristic graph of the pneumoconiotic (silicotic) lung of the peribronchial type. This resembles a capital *W* for one lung, and a double *W* for both lungs (Fig. 25). This graphing becomes spectacular when compared with charts of normal lungs, tuberculous lungs, and lungs manifesting neoplasms.

Normal Lungs (Fig. 24): √ √ Chart resembles a capital *V* with a vertical proximal arm.

Peribronchial Pneumoconiotic Lungs: W W W. (Fig. 25).

Tuberculous Lungs: √‾ W Chart resembles a square root sign in one lung and a capital *W* in the other.

Neoplastic Lungs: √ √ Chart resembles a capital *V* with a vertical distal arm.

We believe that the application of the photometer with the charting suggested would be a convenient way of recording the findings observed in a survey of dust-hazard industries, and that an analysis of such charts would help to differentiate the morbid changes caused by dust irritants in different industries and different sections of the country. It is particularly valuable in differentiating peribronchial pneumoconiosis (silicosis) from other lesions, and since this type of lesion is observed more frequently than the others the photometer is of practical importance. Lastly, such a charting has the advantage of stimulating the roentgenologist to a more careful scrutiny of lung densities.

COMPENSATION

There is no doubt in our mind or in the minds of most observers that moderate deposits of certain mineral dusts cause morbid changes in the lung that can be readily recognized roentgenologically. Whether these changes can be differentiated from changes due to other lesions has been problematical. Miners, buffers, grinders, and employees of some other industries manifest roentgenologic findings not observed in groups of cases not exposed to dust hazards—a pattern of accentuated hilar and linear markings. Very early in his investigation of pneumoconiosis, Pancoast (5) recognized these and discussed their significance as follows:

"Now this appearance of prominent hilum shadows and increased prominence of trunk shadows and linear markings, with or without the faint haze, has in this country at least been designated as the *first stage* of silicosis or pneumoconiosis. There may be some excuse for continuing to call this the first stage, but continued experience with cases of pneumoconiosis developing in various industries has led us to question the wisdom of designating any appearance of pneumoconiosis by any term denoting numerical stages of progress. In this particular instance under discussion, the individual may develop the appearance in a comparatively short period of from one to five years, or not pass beyond it, on the other hand, in fifty years. Moreover, it is not apt to be distinguishable as a stage of the past in the more progressive periods of the condition, and in some industries it may be insignificant or indistinguishable as a stage at all. We prefer to designate the appearance by a term which implies its pathological nature and to call it not a stage but the *perivascular-peribronchial-lymph-node type* or preponderance of the condition.

"The appearance of this type, or preponderance, or stage is by no means characteristic of pneumoconiosis but is found as a result of many other conditions, such as acute bronchitis, chronic bronchial catarrh, bronchiectasis, passive congestion from cardiac decompensation, the infiltrating type of malignancy, and polycythemia. It should not be accepted as an evidence of pneumoconiosis, especially in

medico-legal cases, unless all of the conditions are positively ruled out and the appearance has become present or more marked over a period during which serial examinations have been made. Moreover, even if due to pneumoconiosis, the appearance does not represent an incapacitating degree of the condition in any way, or most certainly not unless it has developed rather rapidly after starting in a dusty occupation capable of producing it. With the very rapid development of pneumoconiosis, one is more apt to find evidences of the interstitial type of fibrosis. We may seem to be unnecessarily verbose in connection with a minor aspect of the condition, but our experience with so many cases of what have seemed unwarranted claims for total disability has forced us into this detailed expression of facts."

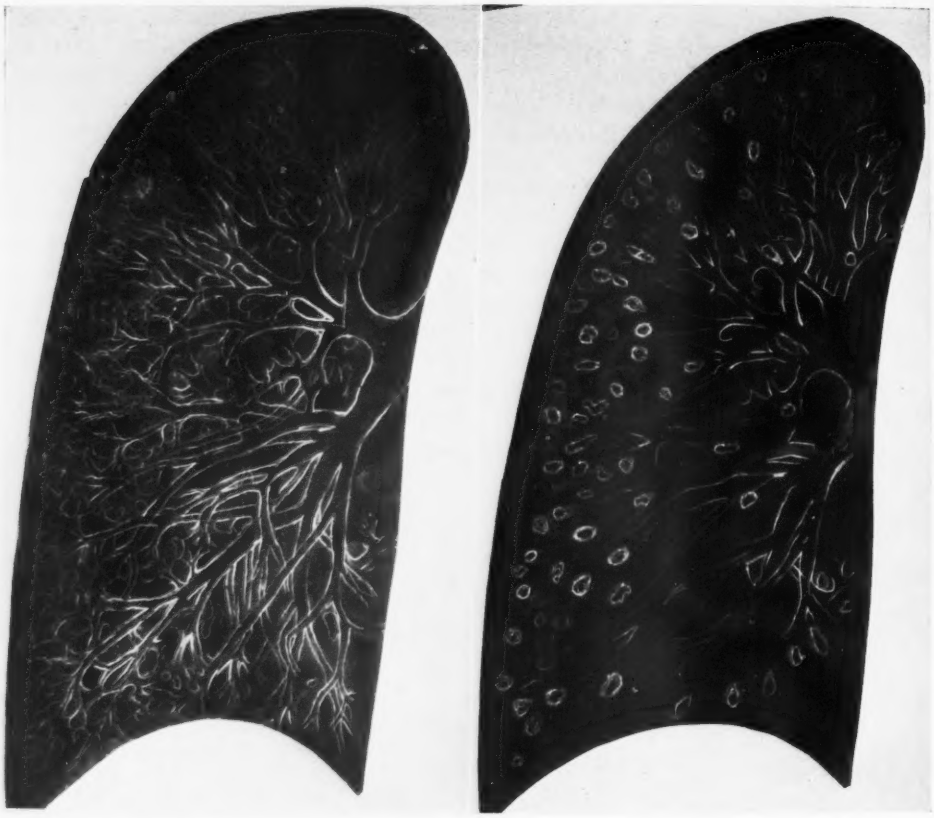


Fig. 14. Fig. 15.

Fig. 14. An accurate tracing of a roentgenogram of a normal lung showing the larger and smaller blood vessels, particularly the fine network in the peripheral third of the lung. These fine vascular loops in the peripheral third correspond with the terminal lobules of the lung and are the anatomical structures on which the collagen is laid down. The alveolar air passages between these loops become choked off by a ball-valve action and dilated air cysts are formed.

Fig. 15. A schematic drawing showing a ring of collagen around these terminal lung lobules. When Figure 15 is superimposed over Figure 14 we have the result illustrated by Figure 4.

This conception of Pancoast's, clearly stated in 1933, is so plain that we can add very little to it. One may wonder why his valuable observation was lost in the shuffle. Our own belief is that it was more or less intentionally lost or mislaid, in an effort to render the roentgenologic diagnosis of pneumoconiosis (silicosis) so simple that the technician, general practitioner, clini-

cian, or health officer need not be forced to depend on an experienced roentgenologist. The commission reporting to the United States Government (1) said:

"A diagnosis may be required of a physician in a plant, in a public health office, in a sanatorium, or in a physician's private office. Sometimes he may have had little experience in the interpretation of roentgenograms of the chest and will be forced to lean heavily on the interpretation of the roentgenologist. The legal aspects of the problem have placed great responsibilities on the medical profession and have been a source of embarrassment. Only the physician who has examined the subject, has obtained an occupational history of adequate exposure to silica dust, and has before him a suitable roentgenogram of the chest, should make the diagnosis of silicosis. The roentgenologist not in possession of these facts can merely state whether the shadows which he sees in the film are consistent with this diagnosis.

"If objective terms descriptive of the type of pathological changes could be generally adopted, material progress would result. The clinician would not have to accept a diagnosis from the roentgenologist, general students of the disease would be able to correlate the findings of various observers, and more accurate definition of roentgenograms would be available for medico-legal purposes. Roentgenograms of the chest, which are notoriously difficult to reproduce as illustrations, could be described in word pictures capable of interpretation in the light of the personal knowledge of observers. An error in diagnosis need not necessarily be passed on to others not in possession of the original film."

Gardner (3), in an elaborate article, says:

"Insufficient exposures to silica may cause a certain amount of exaggeration of the linear shadows cast by the vascular tree, and enlargement of the tracheo-bronchial lymph nodes. These changes are not specific for silica; they may be produced by inhaling other dusts, by infection, and by heart disease. Since all of these factors may be operative upon persons who work in a silica industry, one is not justified in attributing the roentgenologic appearance to silica. Let it be emphasized that without nodulation a diagnosis of silicosis is unwarranted at the present time....."

By and large, the roentgenologists have disregarded Gardner's warning, and have adhered to Pancoast's conception that accentuated hilar and linear markings indicate a peribronchial and perivascular type of pneumoconiosis (silicosis) when other lesions have been ruled out. There seems to be unanimity of opinion that these cases have little clinical significance and are not compensable. Pancoast believed, and we agree with him, that these cases should be considered as pneumoconiosis (silicosis) and attempts made to differentiate them from other linear markings, whereas Gardner, or, at least, the committee reporting to Washington, believed that silicosis should be confined to those cases in which silicotic nodules are so well defined that they are readily recognized roentgenologically. We prefer to meet the issue squarely and admit that this is a type of pneumoconiosis (silicosis) rather than to define pneumoconiosis (silicosis) in terms which include neither this type, which in our opinion should not be compensable, nor acute silicosis, which we regard as more deservedly compensable than the chronic nodular type.

From an intensive study of some two hundred cases diagnosed as pneumoconiosis or silicosis by other roentgenologists and sent to us for study, we were able to compare roentgenologic findings as they develop in different regions and industries, study clinical histories, and correlate the roentgenologic findings with the symptoms of which the patient complains, if and when he does complain. The outstanding revelation of this study was the extensive roentgenologic findings that may occur without the patient having any symptoms whatever; it was not the exception, but the rule. Roentgenograms show lungs shot full of diffuse nodules and pockmarks without the patient even knowing he has a lesion, or, if aware of the roentgenologic manifestations, without being incapacitated in the performance of his work either underground or above ground, within or without a dust hazard. Many of these patients had worked fifteen or twenty years without acquiring clinical symptoms. Even though their lungs were literally studded with collagen, they did not complain of dyspnea, cough, nor expectora-

tion until the roentgenologic nodulation had progressed into massive collagenization, and most of them did not complain relatively few workers incapacitated by lesions acquired and developed in the pursuit of their work. But other controversial

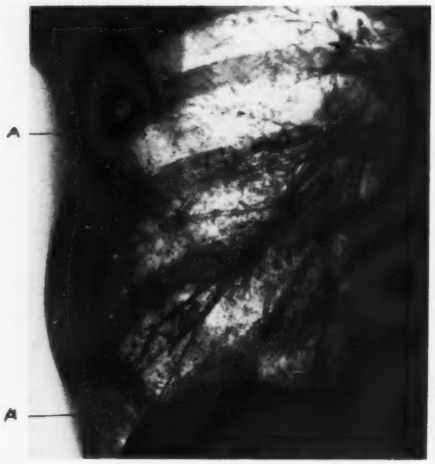


Fig. 16.

Fig. 17.

Fig. 16. Roentgenogram made after the injection of iodized oil, showing the bronchial pattern (*A* and *A'*) on which part of the pockmarks develop.

Fig. 17. Roentgenogram showing a pleural cap at the apex of the lower lobe (*A* and *A'*) beneath which there is massive collagenization. This finding reminds one of rudimentary angel wings folded up on the back of the patient. (*Lent by Dr. Pendergrass, Philadelphia, Pennsylvania.*)

until a gross inflammatory or tuberculous lesion was ingrafted.

The foregoing does not apply to cases of acute silicosis, in which the roentgenologic pattern is a general nondescript haze or cloudiness. Workers exposed to huge amounts of certain dusts containing silica, perhaps silica in combination with some alkaline chemical or gases, develop lesions with great rapidity, passing from an early stage to the terminal stage in a few months, or, at most, in two or three years. Such patients develop dyspnea, sometimes with a dry cough. They die of avascularization of the lung, and these avascular areas may possibly break down because of their poor blood supply. It is to be remembered that in cases of acute silicosis roentgenograms do not show nodules or pockmarks or even areas of massive collagenization.

The question of compensation is vital, and deserving of far greater consideration than it has received thus far. Industry should unquestionably compensate the

questions arise. Some of those which have not been satisfactorily answered are:

1. Should the able-bodied laborer who has a job and wants to keep it be compelled to have an x-ray examination each year and be fired as soon as peribronchial markings or diffuse mottling are manifested on the roentgenogram?

2. Should an employer refuse to hire a man because he has such markings due to previous exposure lest the applicant develop more advanced stages of pneumoconiosis (silicosis)?

3. If a laborer is fired, with compensation which is small in comparison with his wages as a full-time laborer, shall he still receive compensation if he obtains labor in a non-dust-hazard industry?

4. When roentgenologic findings increase without the appearance of dyspnea, cough, or any other symptoms, should the laborer be allowed to work?

5. At what period in the development

of the disease should the laborer begin to receive compensation?

6. Should not those laborers who develop acute silicosis and are rendered totally incapacitated and die within two or three years, be compensated for silicosis even though they do not have the typical nodulation on which the definition is based?

7. Should a tuberculous patient who has worked in a dust-hazard industry but whose roentgenogram shows no evidence of silicosis be compensated for tuberculo-silicosis because of his previous exposure?

It is not our object in this article to answer these questions, but the fact that they are numerous, various, and controversial indicates the necessity of some comprehensive investigation based on factual evidence, both medical and social, so that the incapacitated silicotic victim may be adequately compensated, and unjustified claims may not be a direct burden on industry and thereby an indirect burden on society.

Our investigation reveals that most roentgenologists see occasional cases of pneumoconiosis (silicosis) and welcome the opinion of one making an intensive study of the disease. A few roentgenologists see a tremendous number of cases and are sufficient unto themselves for the diagnosis of cases in their communities. For the convenience of those who rarely see cases, and in the hope of obtaining a comprehensive view of the subject, we solicit the continued co-operation of both these groups. We have begun a registry of silicotic cases, photographing films sent to us, recording the findings, comparing them, and reporting our opinion when returning the films.

This article may tend to be verbose, for it has been most difficult to be convincing without a larger number of illustrations than we have been able to include. In the near future we will have available for distribution scrolls of films showing the four findings of pneumoconiosis (silicosis) and the varying degrees of involvement. Each roentgenogram will have the findings typed and photographed on it, morbid changes and roentgenologic patterns being correlated whenever possible. These films may be studied in conjunction with the article, and will present a far more comprehensive study than we have been able to give in words.

SUMMARY

We are indebted to 24 physicians throughout the country for roentgenograms and autopsy specimens which have made this investigation possible.

It is universally conceded that roentgenograms are important in the diagnosis of pneumoconiosis (silicosis). In our opinion, both worker and employer suffer unless trained roentgenologists are employed for the interpretation of such roentgenograms.

Roentgenograms of subjects who have inhaled certain dusts over a long period of time show findings which are shadows of deposits of morbid tissue composed mostly of collagen. The amount of morbid tissue may be slight or almost great enough to fill the thoracic cage. Since dust is inhaled in both lungs, the distribution is bilateral and relatively symmetrical. Morbid tissue occupies space that would otherwise be filled with air, and casts shadows on the roentgenogram which correspond to it in size, shape, and distribution.

PATTERN APPROACH

Roentgenologic findings will be considered under two main groups: (*A*) characteristic *patterns* on the roentgenogram; (*B*) *regional distribution of densities* on the roentgenogram. Roentgenograms of pneumoconiosis (silicosis) manifest four main patterns which have been discussed in the following order:

1. Accentuated hilar and linear markings.
2. Small white spots on a dark background—nodules.
3. Small black spots surrounded by white rings—pockmarks.
4. A general haze or cloudiness.

1. *Accentuated Hilar and Linear Markings.*—These were said by Pancoast to

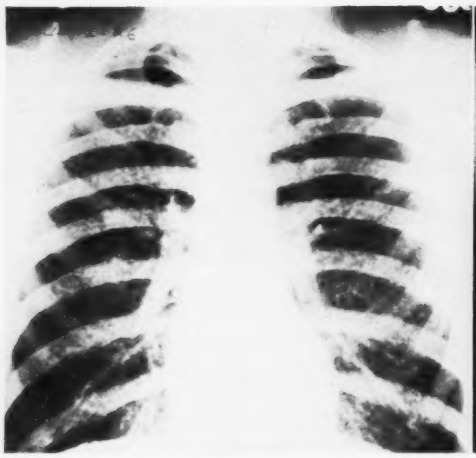


Fig. 18.

Fig. 19.

Fig. 18. Classified by Dr. Smith, of South Africa, as "ante-primary." This roentgenogram shows a relatively early stage of fine bilateral mottling, more marked in both mid-lung fields. The roentgenologic shadows are cast by foreign-body flecks, opaque to both light and x-rays, which have been phagocyted out of the alveoli and deposited in the stroma of the lung, and by small strands of collagen laid down along the branches of the blood vessels and bronchi or laid down as rudimentary whorls. (*Lent by Dr. McArthur, Noranda, Quebec.*)

Fig. 19. Roentgenogram showing a diffuse mottling in both lower lung-fields with large dense shadows in both upper mid-lung fields. The fine mottling of the lower mid-lung field is caused by the same finding described under Figure 18, but the dense shadows above are caused by massive collagenization in these regions. (*Lent by Dr. Pound, New York City.*)

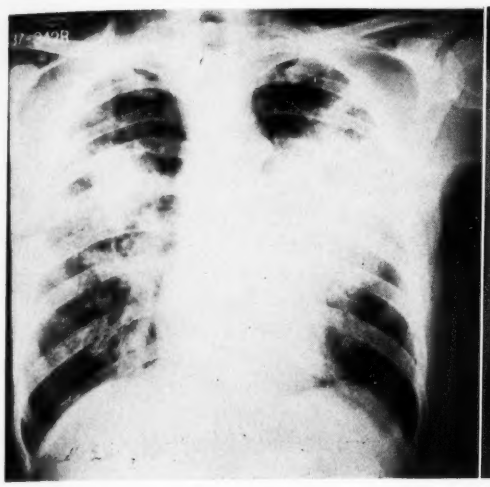
constitute a "perivascular–peribronchial–lymph-node" manifestation of pneumoconiosis. The shadows are due to a deposit of morbid tissue composed of dust-laden phagocytes and collagen laid down like an envelope around blood vessels and bronchi. The phagocytes may be loaded with silica (non-opaque, refracting crystals) or other foreign body flecks (opaque, non-refracting crystals), but in either event are a contributing cause to the accentuated hilar and linear markings.

2. *Nodules—Small White Spots on a Dark Background.*—South African observers gave the name of "nodules" to the small, clear-cut white discs on the roentgenograms of pneumoconiotic (silicotic) patients. These nodules are shadows of whorls of collagen distributed in the parenchyma of the lung, most marked in the mid-lung fields. They may be so small as to be scarcely discernible to the naked eye, or two, three, four, or five nodules may be connected by collagen strands to form a conglomerate nodule.

Nodules have been considered pathognomonic of silicosis, and are the basis of the definition and diagnosis, but nodules may contain a preponderance of opaque, non-refracting flecks (not silica), and we, therefore, use the term "pneumoconiosis" with "silicosis" in parentheses when referring to this lesion.

3. *Pockmarks—Small Dark Spots Surrounded by White Rings.*—This is a roentgenologic finding of importance which we believe has not been recognized before, and it is, therefore, dealt with at length in the article. Pockmarks are shadows of collagen laid down around the terminal blood vessels and bronchi, or collagen around bronchi when viewed on end.

4. *General Haze and Cloudiness.*—A general haze and diffuse cloudiness, bilateral, symmetrical, and not sufficiently dense to obscure the ribs, is the chief roentgenologic finding in acute silicosis. It is caused by the following morbid changes: (1) thickening of the walls of the alveoli; (2) deposits of various types of



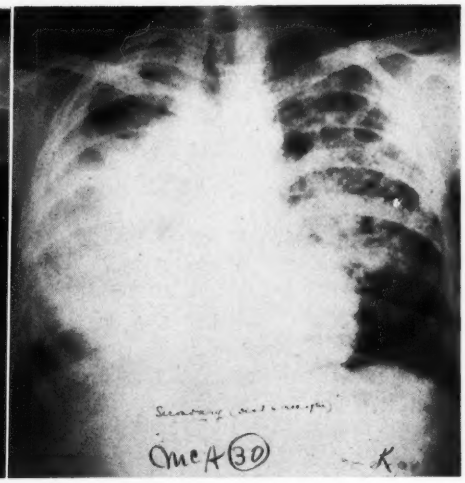


Fig. 20. Fig. 21.

Fig. 20. Shows diffuse mottling in both lung-fields, plus a smooth shadow of collagenization in the upper right mid-lung field. In the left lung one observes diffuse mottling in the lower lung-field, and in the mid-lung field massive collagenization on which infection has been ingrafted. (*Lent by Dr. Corcoran, Scranton, Pennsylvania.*)

Fig. 21. Classified by Dr. Smith, of South Africa, as "secondary with a positive tuberculous sputum." Diffuse mottling of the left lower lung-field. There is a dense, homogeneous shadow in the right mid-lung field due to massive collagenization, probably with some infection. Cavitation of the left upper lung, and a large cavity or localized pneumothorax in the right lung. The patient died six months later. (*Lent by Dr. McArthur, Noranda, Quebec.*)

material within the alveoli; (3) envelopes of collagen surrounding the smaller blood vessels and bronchi.

Massive Collagenization—We have discussed the four patterns in cases of pneumoconiosis (silicosis) in which the disease has progressed sufficiently for the pattern to be well marked but not developed to such a degree that the pattern is obscured on the roentgenogram. Often two or more of the roentgenologic manifestations described may be observed on the same roentgenogram, but one pattern usually predominates.

As the disease progresses, collagen may develop in great quantities, and appear in the following three regions: (1) as a "pawnbroker's sign" in the mid-lung field, seen more frequently on the right; (2) in the pleura at the apex of the lower lobe; (3) in the upper lung-fields.

Ingrafting of Infection—Pyogenic or Tuberculous.—As collagenization increases, the blood supply is impaired and gross infection—pyogenic, tuberculous, or both—may be ingrafted, or, in the case of acute silicosis, develop in the avascular areas. Inflammatory and tuberculous processes do not develop until massive collagenization has occurred. Before infection is ingrafted the markings of pneumoconiotic (silicotic) lungs are not displaced or altered in position, because pneumoconiotic collagen, unlike collagen of tuberculous or inflammatory lesions, does not contract. After infection occurs the trachea may be deviated, the pleura "tented," and the costophrenic angle obliterated.

Cavities and Pneumothoraces.—Cavities may be formed by a breaking down of the tuberculous or pyogenic infection, or a combination of the two. They occur in the upper third of the lung and are of three types: (1) a cavity caused by a tuberculous or non-tuberculous area of collagenization; (2) a spontaneous, localized pneumothorax, usually near the apex; (3) a split pleura, high up on the lateral wall about two or three inches from the apex.

DIFFERENTIAL DIAGNOSIS

The roentgenologic findings of pneumoconiotic (silicotic) lesions must be differentiated from those of numerous pulmonary lesions, and are discussed in the same order observed for the four patterns.

1. *Accentuated Hilar and Linear Markings.*—Those due to pneumoconiosis (silicosis) must be differentiated from those due to pneumonia, post-nasal drippings, chronic passive congestion, neoplastic lesions, and bronchiectasis. Differentiations are not important here from a clinical and compensation standpoint. Roentgenologic differentiation may be made by distribution and location.

2. *Nodules—Small White Spots on Dark Background.*—Nodules of pneumoconiosis (silicosis) should be differentiated from the nodules or mottling of pulmonary tuberculosis, general acute miliary tuberculosis, fungi of yeast infections, neoplastic metastases, and lobar pneumonia. Here again the differentiation may be made by distribution, location, the appearance of individual nodules, and, in doubtful cases, by the aid of clinical histories.

3. *Pockmarks—Small Dark Spots with White Rings.*—Pockmarks due to pneumoconiosis (silicosis) should be differentiated by distribution and location from the pockmarks due to other lesions.

4. *General Haze and Diffuse Cloudiness.*—The general haze is rarely observed in other lesions. In our opinion, the problem in the case of the general haze of acute silicosis is its recognition, not its differentiation from other lesions.

REGIONAL APPROACH

The second approach to the roentgenologic diagnosis of pneumoconiosis (silicosis) is based on the regional distribution of shadows of various morbid changes, regardless of their pattern. These are best observed by viewing the roentgenogram from a distance, squinting the eyes so that one does not see the pattern. For such a study the lung is divided as follows: (1) the apex—the region just below the clavicle; (2) the mid-lung field—between the third and sixth ribs anteriorly; (3) the base—extending from the mid-lung field to the diaphragm.

USE OF THE PHOTOMETER

A casual study of fifty or sixty pneumoconiotic (silicotic) lungs showing accentuated linear and hilar markings was made in 1936 when we first began our study, and these did not then appear to the writers to show any marked or significant changes. When a number of them were placed on light boxes at the same time, however, we observed that there were changes in density in various portions of the lung, particularly in the mid-lung fields adjacent to the hilus. Densities were less circumscribed than in tuberculous lungs.

We began to make lantern slides for further study, and in this connection used the Weston Photometer to determine the amount of light striking the object being photographed, and the amount of light passing through the negative.

It soon became evident that different portions of the chest films being reproduced showed great variation on the dial, and that these densities varied from densities of normal lungs, tuberculous lungs, or lungs showing other morbid changes. A recording of densities in the aforementioned fifty or sixty roentgenograms was made. The resulting numbers had no real significance until a charting and a graphing were made. Just as the charting of temperatures is impressive in diseases such as typhoid, malaria, acute infection, etc., so a charting of densities in lungs exhibiting increased hilar and linear markings became most illuminating. Five readings were made of each lung and charted on graph paper. Typical readings of a normal lung gave a capital *V* with a proximal vertical arm. Typical readings of pneumoconiotic (silicotic) lungs showing increased markings gave a capital *W* for each lung or a double *W* for both. Typical readings of tuberculous lungs gave a square root sign for one lung and a *W* for the other. Typical

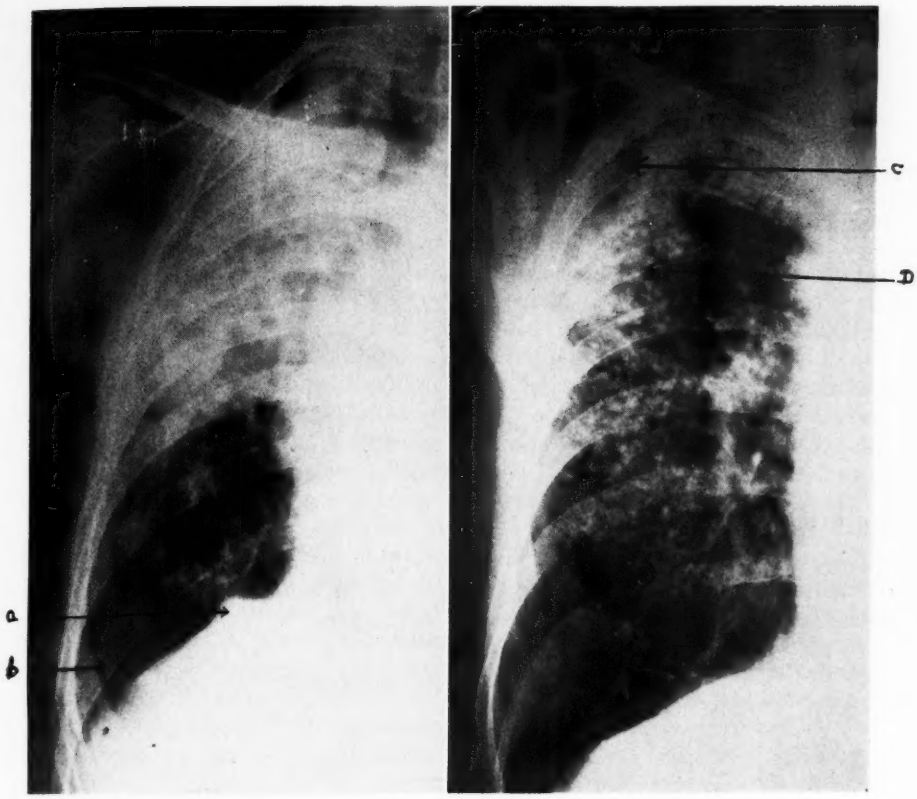


Fig. 22-*A*. Fig. 22-*B*.

Fig. 22-*A*. Exposure to silica dust as a sweeper; no symptoms. Roentgenogram shows (*A*) tenting of the right diaphragm due to a cicatricial contraction of a thickened inflammatory pleura in the fissure of the lung, and (*B*) obliteration of the costophrenic angle. There is also a deviation of the trachea to the right. These findings indicate that an old infectious fibroid lesion has been ingrafted upon the silicosis, if the silicosis exists. If it exists, it has been so obscured by the infection that the diagnosis is rendered difficult and doubtful. (*Lent by Dr. Cook, Worcester, Massachusetts.*)

Fig. 22-*B*. Exposure to silica. Roentgenogram showing split pleura high up in the axillary line at the right apex (*C*). No retraction of the trachea. Pockmarking adjacent to the pleura shown very distinctly (*D*). (*Lent by Dr. Klein, Perth Amboy, New Jersey.*)

readings of neoplastic lungs gave a capital *V* with a vertical distal arm for each lung.

We believe that this accurate way of measuring densities, and the use of the charting suggested, leads to an unbiased interpretation of lung densities and would be invaluable in making surveys of dust-hazard industries. It would enable the roentgenologist to differentiate the increased markings of pneumoconiosis (silicosis) from those due to other lesions, and these chartings, with a study of the pattern, would aid him in recognizing and differentiating the morbid changes due to dust in different communities and industries.

COMPENSATION

Most observers agree with Pancoast that accentuated hilar and linear markings are evidence of pneumoconiosis (silicosis) when other lesions have been ruled out, and that the disease in this stage should not be compensable. Gardner, however, and the committee reporting to Washington regarded a diagnosis of silicosis unwarranted unless roentgenograms showed definite nodulation. This would exclude

the pattern of accentuated markings and also acute silicosis.

Our examination of about two hundred roentgenograms from various industries and sections of the country led to one outstanding revelation—that subjects do not complain of dyspnea, cough, or expectoration in pneumoconiosis (silicosis) until the development of massive areas of collagenization or areas of collagenization on which infection, either tuberculous or pyogenic, has developed. They may work 15, 20, or 30 years without symptoms, though their roentgenograms show shadows of extensive morbid tissues.

In the case of acute silicosis, which develops and terminates within two or three years, the subject does have symptoms, and dies of avascularization of the lung. We believe that such cases should be compensated, though they do not now come within the definition of silicosis.

The question of compensation is vital and is deserving of far more consideration than it has received thus far. It is not our object to discuss the questions which were brought up in the article in regard to it, but to emphasize the necessity for a comprehensive investigation based on factual evidence both medical and social, so that the incapacitated silicotic victim may be adequately compensated, and unjustified claims may not be a direct burden on industry.

APPENDIX

Fundamental Principles Underlying X-ray Diagnosis in General.—It is well known that x-ray has ability to penetrate objects opaque to light. The ray is obstructed by objects in proportion to their radiological density, or inversely in proportion to the penetration of the x-ray. For example, in the human body rays are obstructed by bullets, bones, and kidney stones in proportion to their density, the bullet obstructing the rays to a greater degree than the bones. Bones, on the other hand, are more opaque than muscles and soft tissues and, therefore, obstruct a larger proportion of the x-rays.

Roentgenograms were formerly referred to as "shadow graphs" or "skiagraphs," and are sometimes likened to silhouettes, but the roentgenogram is more than a silhouette for it records the opaqueness of objects.

In a well-exposed roentgenogram with a properly selected penetration of the ray, bones, muscles, blood, and fat will cast shadows of varying intensity, the bones obstructing the rays to the greatest extent and, therefore, appearing as the lightest area on the roentgenogram. Other less solid objects appear light in proportion to their solidity in the body. More opaque substances such as bismuth, the iodide oils, and dyes are often injected or ingested into the hollow cavities to aid in x-ray diagnosis. Areas of increased solidification in the body always appear as areas of lightness on the roentgenogram, whereas regions of the body containing air (accessory sinuses, mastoids, lungs, etc.) do not obstruct the rays as much as the soft tissue which surrounds them, and appear as blacker areas. While these areas of increased density in the body appear as varying degrees of light areas on the roentgenogram, we will translate these shadows into areas of anatomic and pathologic density.

There is about equal division of opinion as to whether roentgenograms should be reproduced as negatives or positives. When describing the roentgenographic print, or positive slide, the term "density" applied to the roentgenogram corresponds with pathologic density in the lung. For those who prefer to observe the negative findings, or black on white, the terms will be reversed. In this article, illustrations are given in both the negative and positive.

Special Principles Applicable to the Diagnosis of Chest Lesions.—The lung contains a far greater amount of air than any other organ of the body, so even the soft tissues of the lung, being in contrast to air, are shown on the roentgenogram as areas of diminished density, which, translated into anatomic structures, means areas of increased density, the degree of increase

being in direct proportion to the degree to which they obstruct the x-rays. Therefore, these soft tissues of the lung appear as definite shadows on the roentgenogram, causing a characteristic pattern in the normal lung. Soft tissues of the same density occurring in airless regions of the body would not be observed on a roentgenogram.

Almost all morbid tissues in the lung increase its solidity, and are manifested on the roentgenogram as areas of diminished density, which, after translation, we refer to as areas of increased density, whereas morbid accumulations of air in the lung reduce its solidity and are manifested as areas of diminished anatomic density, as in the case of emphysema and over-distention of the lung as seen in cases of non-opaque foreign bodies. Thus different morbid processes as they occur in different portions of the lung obstruct or increase the transmission of x-rays, causing peculiar patterns that are more or less characteristic of the morbid lesions. The more air there is in the lung the more clearly are the disease areas shown, and as the lung collapses the tubercles or nodules become obscure.

We remember well a controversy which took place in the early days of pulmonary roentgenology, concerning whether or not a miliary tubercle would cause an x-ray shadow. Cohn made an x-ray examination of a small section of a tuberculous lung which he knew to contain miliary tubercles, and this section which was, of course, collapsed, failed to show the miliary tubercles on the roentgenogram. He, therefore, maintained that it would be much more difficult to show these through the muscles of the thoracic cage; but he overlooked the fact that the tubercle in the collapsed lung was surrounded by tissue of relatively the same density, whereas in the inflated lung during inspiration it was shown in contrast to air. Roentgenograms of autopsy specimens soon showed that tubercles were not visible in collapsed lungs, but that when the same lung was inflated they could be identified and readily counted. In life, the same thing is true. Miliary tubercles and the normal markings are evident during inspiration, but they are obscured when the lung is even partly collapsed by pneumothorax. Thus degrees of inflation, or the depth of inhalation, are important factors in x-ray technic. Ernst (7), of St. Louis, in discussing densities in silicotic lungs, illustrated clearly the difference between roentgenograms made during inspiration and those made during expiration.

When a lung is prepared in the conventional way for a microscopic examination, it contracts and is further diminished in size by fixation, so that a section of the lung that extends all the way from the hilus to the pleura can be mounted on a single microscopic slide. The contraction of the microscopic section is due largely to the elimination of air, which causes the alveoli and bronchi to contract and appear much smaller than in the living subject. Thus the microscopic findings, particularly the size and shape of the alveoli and bronchi, cannot be compared with those same structures in the living, functioning lung.

In the normal lung, where the walls of the bronchi are thin, the air in them cannot be readily differentiated from the air in the adjacent alveoli, but in the typical pneumoconiotic (silicotic) lung there is a thick envelope of collagen surrounding the bronchi, and the air within the two may be differentiated on the roentgenogram. The larger and medium-sized bronchi radiate from the root of the lung and one is able to trace the course of the air they contain. As viewed longitudinally, such a bronchus appears to be like a hollow tube, while a bronchus that projects directly anteriorly or directly posteriorly looks like the end of a pipe, or a circle surrounded by increased density. These bronchi, seen on end, are occasionally observed in the normal lung, but they become much more evident in the pneumoconiotic lung and account in part for the "pockmarks" on the roentgenogram.

TECHNIC OF CHEST X-RAYS

While each roentgenologist has his own personal technic there are certain funda-

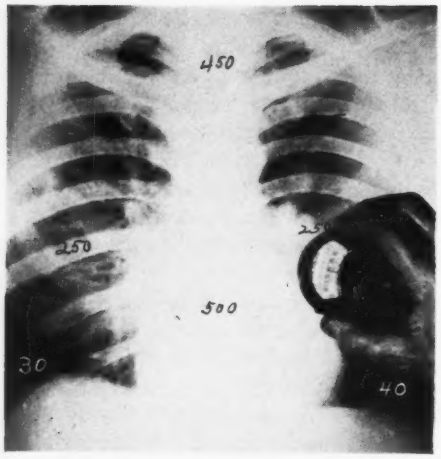


Fig. 23. Photometer held over roentgenogram to detect the increased lung densities with mathematical accuracy. This roentgenogram shows increased lung density in the mid-lung field.

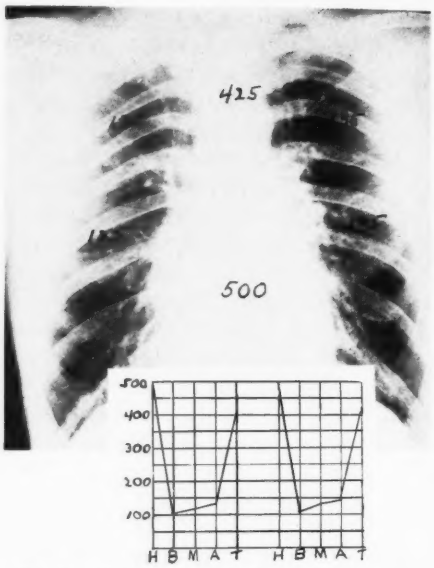


Fig. 24. Roentgenogram of a normal lung showing the lung densities as observed by the photometer. When readings are recorded on a chart, the chart gives a capital *U* for each lung, or a double *U* for both lungs.

mental principles to which it seems wise to adhere in the making of chest roentgenograms.

Distance.—The target–film distance of six feet has been adopted with a fair uniformity. Distances of eight or ten feet may be used without markedly increasing the detail or diminishing the distortion, and distances of three to four feet may be used, but these do increase distortion although they diminish the time, which in every roentgenogram varies with the square of the target–film distance. We believe that the standard distance of six feet should be adopted wherever possible.

Penetration.—The penetration of the rays from the more modern apparatus is indicated by kv.'s (kilovoltages). A kilovoltage with a given milliamperage gives uniformly the same penetration with the same apparatus, but the kilovoltage on one machine cannot be compared with the kilovoltage on another machine, particularly if one is of the valve tube rectification type. In studying films of the chest one can determine whether they are under-penetrated and under-exposed by noting whether or not the ribs are visible through the heart. We believe that in a satisfactory roentgenogram the ribs should be just discernible through the heart. If they are not discernible, the roentgenogram is underexposed because of insufficient time or penetration; but if the ribs show too distinctly, the structural details of the smaller normal markings of the branching of the blood vessels near the periphery will be wiped out. With the correct exposure, the ribs are just barely visible and the linear markings are shown at the periphery of the lung. Each roentgenologist or his technician should get as great an amount of variation, or gradation of shadow in the chest, as is obtainable with the apparatus used.

Time.—The time of exposure should be sufficient to give the detail just described. If the apparatus used enables the roentgenologist to make a properly exposed roentgenogram in one-twentieth of a second or less, the time factor is ideal. One-twentieth of a second is chosen instead of one-tenth because one-twentieth is sufficiently rapid so that the motion of the heart does not blur that portion of the lung which lies adjacent to it, and with

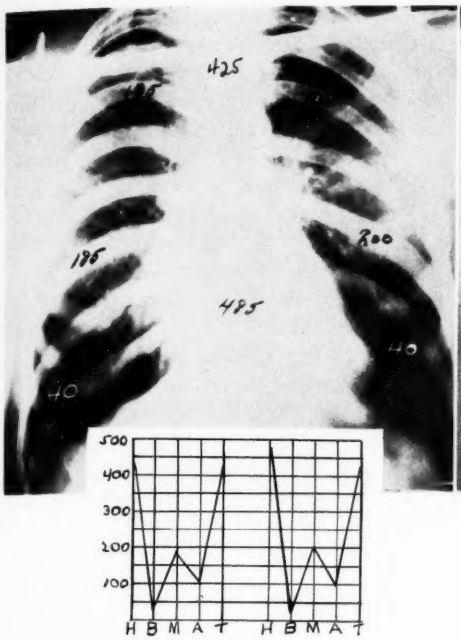


Fig. 25.

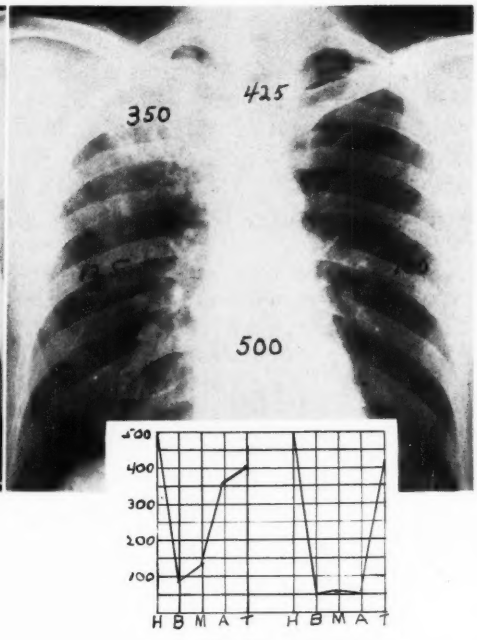


Fig. 26.

Fig. 25. Photometric readings recorded on a roentgenogram showing the peribronchial-perivascular type of pneumoconiosis, with increased ventilation at the bases and diminished ventilation in the mid-lung fields. When readings are recorded on a chart, the chart gives a typical capital *W* for each lung, or a double *W* for both lungs.

Fig. 26. Photometric readings recorded on a roentgenogram showing pulmonary tuberculosis of the right upper lobe. When the readings are charted, the chart shows a square root sign on the affected side, and a capital *U* on the normal side.

such an exposure or less the left border of the heart is clear-cut on the roentgenogram. When an exposure of one-tenth of a second is used the left border of the heart is blurred if the heart is caught in the stage of contracting or relaxing, but if the exposure happens to catch the heart in complete diastole or systole, the motion is *nil*. Actually the heart is observed to be at rest in about one-half of the films made with one-tenth of a second exposure. If it is necessary to make an exposure of one-fourth of a second, there is so much motion to the heart and the adjacent lung that it is just as well to increase the time to one-half of a second or more, and in order to obtain a film of proper density increase the contrast by using less penetration.

As stated before, the ideal time is one-twentieth of a second or less. Shorter exposures or less penetration may be used for special regions in the chest which are over-ventilated, or in special cases involving the bases of the lungs—as in emphysema. On the other hand, when the ventilation of the lung is displaced by a large mass of morbid tissue, the detail in these dense portions of the advanced lesions may be shown, as Pancoast and Pendergrass (2) pointed out, by the use of the Potter-Bucky diaphragm. This technic will bring out pneumoconiotic (silicotic) nodules which would otherwise be obscured by consolidation or interstitial deposits of collagen, but we wish to emphasize the fact that the Bucky technic must be used only as a supplementary procedure for the examination of advanced lesions.

Study of the Roentgenogram.—Roentgenograms should be studied by properly

transmitted illumination and care should be taken to eliminate all extraneous light, particularly the reflected light from behind the observer. A rheostat, or some device for increasing or diminishing the intensity of the light in the illuminating box, is essential. The use of the fluoroscope for the study of pneumoconiosis (silicosis) is relatively limited because it affords no permanent record with which to compare subsequent examinations. It is of value in studying the excursion of the diaphragm as this can be recorded on a translucent paper behind the screen, but in all controversial cases it is better to make two roentgenograms with the patient in the same position, one during inspiration and the other during expiration.

Diagnostic Value of Paper Roentgenograms Compared with Films.—Some thirty odd years ago, we suggested the use of paper instead of plates which presented storage and breakage problems, but it soon became apparent to us that paper roentgenograms were less satisfactory, and when the film came into vogue and the problems of breakage and storage were solved, the advantages of paper were eliminated. Recently the use of photographic paper in place of films has been advertised and widely advocated by a firm of commercial radiographers who seek the business of making surveys of employees in dust-hazard industries. They offer to make paper roentgenograms at a contract price lower than is possible for the roentgenologist using individual films or even rolls of films. This company submitted to the New York Society of X-ray Economics samples of paper and film roentgenograms, both made on the same group of tuberculous and pneumoconiotic (silicotic) patients.

On the paper roentgenograms the ribs, the outline of the heart, calcified glands, and grosser lesions were visible, but the smaller lesions, particularly the smaller tubercles, nodules, and linear markings could not be seen. This would have led the roentgenologists into serious errors of omission in their interpretations. The diagnostic value of films made of the same patient was so much greater that there was literally no comparison. The use of paper roentgenograms for silicosis surveys was particularly unfortunate, and an expensive economy for the industrialists. It is well known that in the early stages of the disease clinical symptoms are absent, and that one must depend upon satisfactory roentgenograms for diagnosis. The paper roentgenogram fails to show the early findings clearly, and hence an industry using them would undoubtedly accept as free from pneumoconiosis (silicosis) employees who would later develop symptoms, become claimants, and represent a compensation burden. The compensation of one individual so accepted would be far in excess of the price difference between paper and film roentgenograms.

REFERENCES

(1) Roentgenological Appearances in Silicosis and the Underlying Pathological Lesions. U. S. Public Health Reports, August, 1935.

(2) PANCOAST, H. K., and PENDERGRASS, E. P.: Roentgenological Aspects of Pneumoconiosis and its Medico-legal Importance. Jour. Indust. Hyg., **15,** 117–135, May, 1933.

(3) GARDNER, L. U.: Pathology of the Pneumoconioses. New York St. Jour. Med., **36,** 1377–1381, Oct. 1, 1936.

(4) DELAFIELD, F. A., and PRUDDEN: A Handbook of Pathological Anatomy and Histology. William Wood & Co., New York City, 1904.

(5) PANCOAST, H. K., and PENDERGRASS, E. P.: Roentgenologic Aspect of Pneumoconiosis and its Differential Diagnosis. Jour. Am. Med. Assn., **101,** 587–591, Aug. 19, 1933.

(6) GARDNER, L. U.: Pathology of So-called Acute Silicosis. Am. Jour. Pub. Health, **23,** 1240–1249, December, 1933.

(7) ERNST, EDWIN C.: Roentgenologic Aspect of Silicosis. RADIOLOGY, **30,** 285–293, March, 1938.

A VISUALIZATION STUDY OF FIBROTHORAX: IDENTIFICATION OF THE CARDIOVASCULAR STRUCTURES[1,2]

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IN patients having massive pulmonary fibrosis, or "fibrothorax," the conventional roentgenogram fails to reveal the essential features of the thoracic cardiovascular system. However, visualization of these structures is now possible, for, recently, we have developed a method for the visualization of the chambers of the heart, the pulmonary circulation, and the great vessels (1, 2), and have reported examples of its value in the normal individual (3) and in patients having heart (4) and lung (5) disease. It has been possible to see the cavity and wall of each chamber, the interventricular septum, the pulmonic and aortic valves, the pulmonary artery and wall, the entire pulmonary circulation, and the thoracic aorta with its wall and the branches from the arch.

In this paper we wish to illustrate the value of this method in a patient in whom there was displacement and obscuring of the heart by pulmonary and pleural fibrosis and to show that it is now possible to visualize the cardiovascular structures as well as the esophagus and the tracheobronchial system.

Method and Roentgenographic Technic.—Visualization studies were made in the lateral, the left anterior oblique, and the frontal positions. By using a rapid stereoscopic cassette shifter, we were able to make two exposures per injection and, thus, to visualize both sides of the heart and the large vessels. The right cephalic vein, which measured 8 mm. in diameter when distended, was used for each injection since it was the only large vein available; the injections were made through a special 12-gauge needle-stopcock unit. Before injection of the contrast substance, the arm-to-pulmonary and the arm-to-carotid sinus circulation times were determined and found to be three and eight seconds, respectively. Thus it was possible to learn the time of the arrival of the contrast substance in both the right side of the heart and the pulmonary arterial tree, and the left chambers and the aorta. The patient was then seated before the cassette and 35 c.c. of a 70 per cent solution of diodrast injected in two seconds. In addition to the usual wave of heat felt immediately after injection, the patient developed a small area of edema of the lower lip which was promptly relieved by 0.2 c.c. of epinephrine, and subsequently was prevented by premedication with this drug. Immediately after injection, the patient was allowed to go home. The absence of detectable injury to the vein was proven by careful examination and by its repeated use (1, 2).

For the frontal views, the following exposure factors were used for this patient in whom the postero-anterior diameter of the chest was 7.5 inches; milliamperage, 300; distance, 72 inches; exposure, one-twentieth second; 66.2 kv. (peak) for the control film (Fig. 1) and 80.5 for the contrast films (Figs. 4, 5, and 6). In order to secure good contrast and detail, it was found necessary to overpenetrate the films showing the contrast substance as in bronchography (Figs. 4, 5, 6, 8, 9, 11, and 12). For the left anterior oblique projection, the same factors were used except that the voltage was increased to 97 kv. (peak) (Figs. 7, 8, and 9). The lateral films (Figs. 10, 11, and 12) were taken at 100 ma., distance 72 inches, exposure one-fifth second, and 90 kv. (peak).

[1] This is one of a series of papers contributed by friends and former pupils of I. Seth Hirsch, M.D.

[2] This investigation was aided by a grant from the Department of Medical Research of the Winthrop Chemical Company, Incorporated.

Case Report.—M. R., a 36-year-old Italian housewife, was first seen in January, 1938, because of cough and hemoptysis. and her height 4 feet 9 inches. Examination of the chest revealed deviation of the trachea to the right and dullness over the

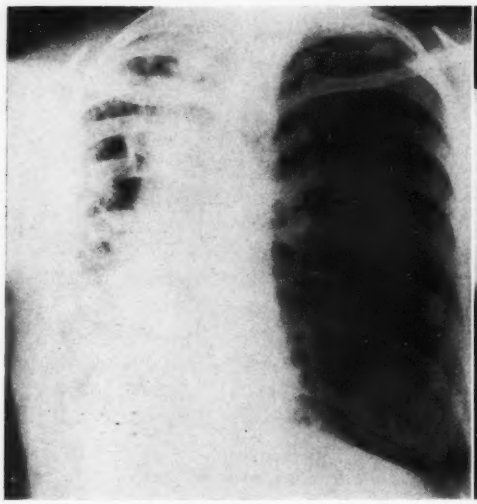

Fig. 1.

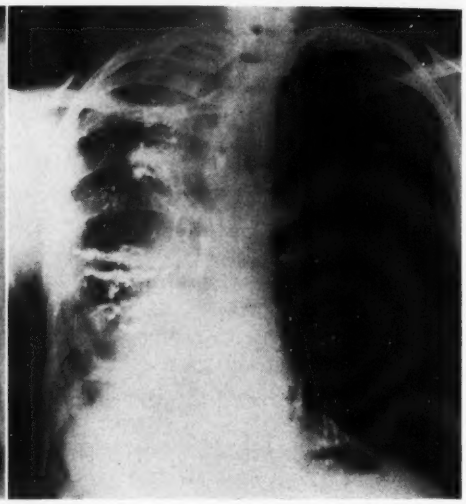

Fig. 2.

Fig. 1. Conventional roentgenogram. Bilateral apical fibrosis with multiple small excavations in right upper lung-field. Fibrosis of the entire right lung, particularly apex and base, causing displacement of trachea, mediastinum, and heart. The cardiac outline cannot be seen. Minute opacities in right upper lung-field due to retained lipiodol.

Fig. 2. Frontal bronchogram. Note curvature and widening of trachea with its bifurcation in the right side of the chest. Sacculated bronchiectasis throughout the right lung.

Ever since the occurrence of whooping cough at the age of four years, she had had episodes of coughing, termed "bronchitis," which were never incapacitating. After the birth of her second child, in 1925, she was examined and found to have a "lung condition," but no roentgenogram was made. Except for an occasional "chest cold" she considered herself well until a month prior to her first visit, when she had "pleurisy" in the right lower axillary region, and, in addition, cough, scanty mucoid expectoration, blood streaking on two occasions, and an hemoptysis of 30 c.c. once. There was considerable lassitude and a loss in weight of ten pounds. She never complained of dyspnea although she occasionally reported wheezing.

Physical examination at that time showed the patient to be well developed and nourished and in no discomfort. She was afebrile. Her weight was 83 pounds upper two-thirds of the right side of the chest, front and back. The breath sounds were bronchovesicular in character and were accompanied by numerous moist râles, many of which were consonating. Occasionally, wheezing was heard over the right anterior side of the chest. The heart was displaced into the right side of the thorax; the sounds were of good quality and no murmurs were present. The blood pressure was systolic 120, diastolic 85. There was no clubbing of the fingers or toes.

These signs and symptoms, in addition to the presence of multiple excavations in the right upper third of the lung-field (Fig. 1) and the apical fibrosis on the left side, suggested active pulmonary tuberculosis. The chronic nature of the process was indicated by the displacement of the trachea and the heart to the right and by the pleural thickening at the base and apex.

However, after six concentrated specimens of sputum proved negative for tubercle bacillus, active tuberculosis seemed unlikely, and it was decided to investigate further by bronchoscopic examination and, later, bronchography. Bronchoscopy revealed narrowing of the right main bronchus and the presence of a moderate amount of mucoid secretion. The bronchogram (Fig. 2) showed marked deviation of the trachea to the right with displacement of the bronchi, and also sacculated bronchiectasis involving the right upper, middle, and lower lobes; a lateral film showed the sacculations in the upper lobe more distinctly. The esophagus lay to the right of the thoracic spine which it crossed at the level of the eleventh vertebra to enter the stomach. In the roentgen kymograph (Fig. 3),[3] the ventricular and pulmonary arterial waves to the left of the spine could be seen clearly, whereas the aortic waves to the right of the spine were barely detectable and other waves were absent. The electrocardiogram was normal except for high voltage; the Wassermann test was negative.

During the past year of observation the patient has been fairly well. Her weight has increased to 96 pounds and she has had only one recurrence of cough and expectoration of blood-streaked sputum. Serial roentgenograms have remained unchanged and the sputum has been negative for tubercle bacillus on two more examinations.

CONTRAST ROENTGENOGRAPHIC STUDIES

Postero-anterior Position.—Figure 4 was made one second after the beginning of injection. The right cephalic, axillary, and subclavian veins are filled with the contrast substance and there is reflux into the basilic vein. The right subclavian vein is kinked and foreshortened as a result of the shift of the mediastinum and its contents to the right. The innominate vein lies to the right of the trachea and follows its contour. The upper left arrow indicates the junction of the right and left innominate veins forming the superior vena cava, which courses downward to the right of the trachea and the right main bronchus; it appears irregular on its medial side. The right auricle is indicated by two small arrows and the auriculo-ventricular groove by a large arrow.

Fig. 3. Roentgen kymograph. Pulsation of the left ventricle, the left pulmonary arterial tree, and the aorta only are discernible. Note absence of waves in the lower right lung-field.

In Figure 5, made at three seconds after the start of injection, the right innominate vein and superior vena cava still show faint filling, while the right auricle, the right ventricle, and the pulmonary arterial tree are opaque. The lower arrow points to the diaphragmatic border of the right ventricle. The auricle and the ventricle appear to be rotated slightly toward the left. The pulmonary conus is not well outlined but lies immediately below the left pulmonic sinus which is denoted by the arrow over the spine. The pulmonary artery courses backward and divides into right and left branches. The dwarfed right trunk which terminates in a few small branches is indicated by the upper right arrow. The left trunk and its major divisions appear more prominent and larger than the normal.

[3] Obtained through the courtesy of I. Seth Hirsch, M.D.

At eight seconds (Fig. 6), the left ventricle and the thoracic aorta are well outlined and are found to be displaced far to the right. The interventricular septum is indicated by the lower left arrow and the aortic sinus and ascending aorta by the upper right arrow. The pulmonary veins can be seen entering the left hilum, whereas

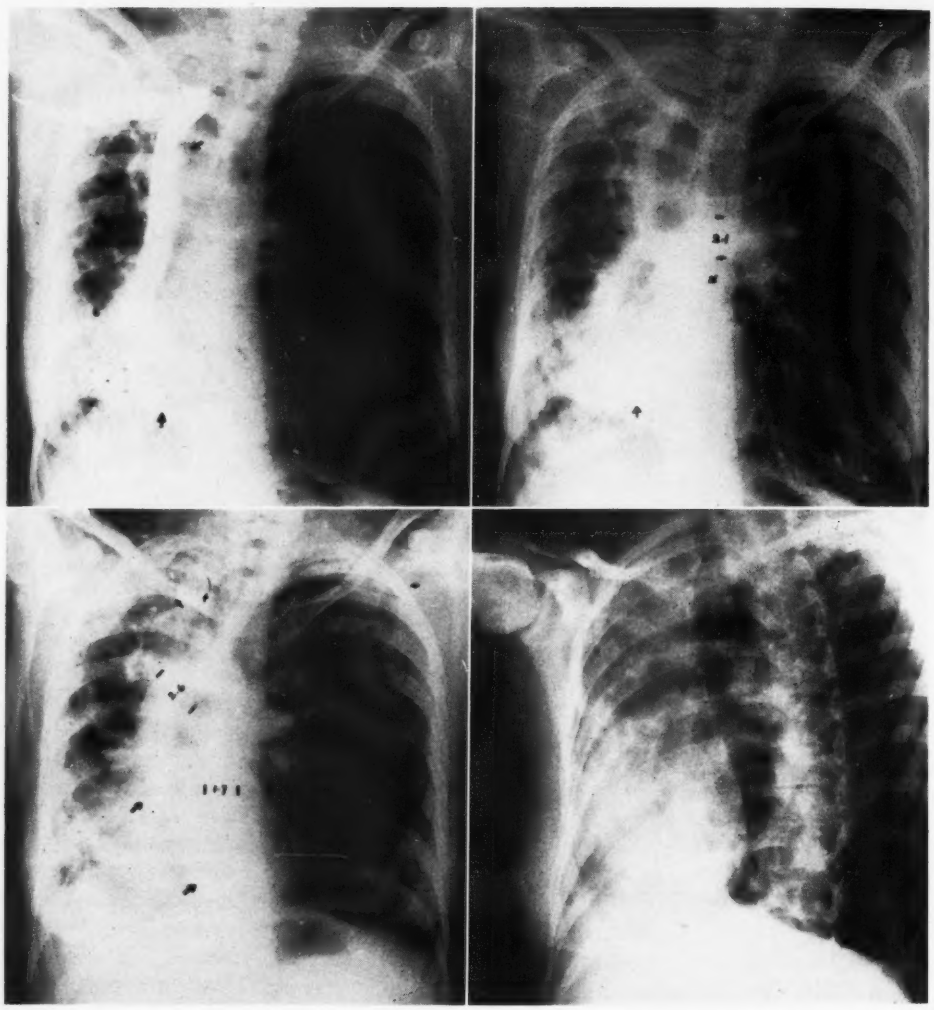

Fig. 4 (*upper left*). Contrast roentgenogram, frontal view. Note kinking of the right subclavian vein and displacement of innominate vein and superior vena cava. The right auricle (denoted by darts) is beginning to fill. The upper left arrow indicates junction of innominate veins; lower left arrow, auriculo-ventricular groove.

Fig. 5 (*upper right*). Contrast roentgenogram. The lower right arrow points to diaphragmatic contour of right ventricle, and the arrow over the spine indicates the left pulmonic sinus. The upper right arrow denotes dwarfed right branch of the pulmonary artery. Note paucity of blood vessels to right lung and rich vascularity of left lung.

Fig. 6 (*lower left*). Contrast roentgenogram. The left ventricle and aortic arch are displaced into right side of chest. Lower right arrow points to interventricular septum; the arrow above it to right aortic sinus. The right subclavian, the right common carotid, and the left axillary arteries are indicated by darts. Note pulmonary veins at left hilum.

Fig. 7 (*lower right*). Conventional roentgenogram in the left anterior oblique position (35 degrees). Note obscuring of right cardiac border.

these vessels cannot be identified on the right side. The innominate artery which arises at the aortic arch may be seen dividing into the right subclavian and the right branches from the arch are opaque. The lower right arrow rests on the interventricular septum and points to the left ventricle; the auricle lies above and behind. The

Fig. 8.

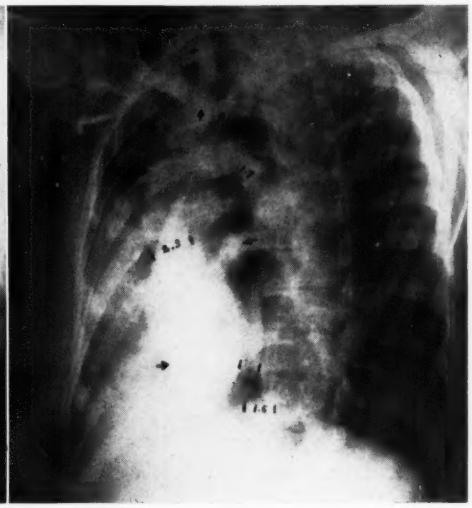

Fig. 9.

Fig. 8. Contrast roentgenogram. The superior vena cava and tributary veins, the right auricle, and the pulmonary artery and its main branches are opaque. Small arrows enclose auricle; larger arrows indicate atrophied branches of right main artery.

Fig. 9. Contrast roentgenogram. The left arrow lies on a left pulmonary vein; the right arrow indicates the left ventricle and interventricular septum; the ventricular wall is enclosed by parallel lines. The entire thoracic aorta is opaque. Darts indicate three main branches from the aortic arch and the major subdivisions of the innominate artery.

common carotid arteries which are indicated by small arrows. High in the left axilla is the axillary artery marked by an arrow.

Left Anterior Oblique Position.—Roentgenoscopy showed that the best view was obtained with a rotation of 35 degrees. In Figure 8, made at two and one-half seconds, the superior vena cava and its tributaries can be seen emptying into the right auricle, which is bracketed by small arrows. The right ventricle which apparently is in contraction is not opaque whereas the pulmonary artery and its left branch are well outlined. The shrunken upper and lower branches from the right main trunk, which are poorly defined, are denoted by arrows.

Figure 9 was made at eight seconds. The left pulmonary veins, the left auricle and ventricle, and the thoracic aorta with the aorta, which can be followed from its origin to the diaphragm, is normal in size, but the aortic arch is displaced to the right and is more rounded than usual. The three vertical arrows indicate the innominate, the left common carotid, and the left subclavian arteries. The innominate artery divides into the right common carotid artery which can be followed to the neck and the right subclavian artery and its continuations which are visible as far as the arm. The corresponding arteries on the left side also can be seen.

Lateral Position.—In Figure 11, made at two and one-half seconds, the right auricle, the right ventricle including the pulmonary conus, the pulmonary artery, and the left main branch with its subdivisions are opaque. The rounded density above the lowest arrow is the right auricle; behind

and above it lies the left auricle (posterior arrow), and in front of it the right ventricle, which is indicated by the arrow next to

Fig. 10. Conventional lateral roentgenogram.

the sternum. The pulmonary conus is embraced by parallel lines as is the larger pulmonary artery above it. The left branch of the pulmonary artery breaks up into superior and inferior divisions whereas the right branches are ill-defined.

The left auricle, the left ventricle, and the thoracic aorta are opaque in the roentgenogram made at eight seconds (Fig. 12). The vertical arrow indicates the left ventricle which is not clearly outlined. The ascending aorta, including one of the aortic sinuses, and the descending aorta are filled with opaque medium and are normal in size and contour. The transverse aorta is obscured by the pulmonary disease.

DISCUSSION

The conventional methods of investigation of the chest in fibrothorax, consisting of ordinary roentgenography assisted by contrast visualization of the bronchi, the esophagus, and the pleura, roentgen kymography, planigraphy, bronchoscopy, and sputum examination, permit the diagnosis of fibrothorax and recognition of the etiology and gross anatomical changes. But they fail to disclose the location and the structural and functional state of the heart, the great vessels, and the pulmonary circulation because of the obscuring pulmonary disease. Consequently, it is impossible to recognize pathologic conditions in the cardiovascular structures in the thorax by ordinary technic.

In the present case, conventional methods have established the diagnosis of fibrothorax and bronchiectasis and have indicated the probable rôle of tuberculosis in the etiology because of the bilateral apical involvement. The extensive pulmonary fibrosis and the displacement of the heart and the other mediastinal structures into the right side of the thorax are shown clearly in the usual roentgenogram (Fig. 1). Bronchoscopic examination revealed stenosis of the right main bronchus, and excluded foreign body and neoplasm as etiologic factors. The presence of extensive sacculated bronchiectasis involving the entire right side is shown in the bronchogram (Fig. 2). The cardiac silhouette, however, is completely obscured in the ordinary roentgenogram. Roentgen kymography (Fig. 3) permitted recognition of segments of the left ventricular border, the aorta, and the left pulmonary vessels but failed to reveal the right border of the heart and the right pulmonary blood vessels.

By means of contrast roentgenography of the cardiovascular system, it has been possible to resolve the unintelligible densities in the customary roentgenogram into their component parts. The four chambers of the heart, the great vessels, and the pulmonary circulation become visible, and changes in the size, shape, and position can be detected. The degree of displacement of the mediastinum is indicated by the kinking of the right subclavian vein and by the location of the superior vena cava (Fig. 4) which can be followed from its origin in the junction of the innominate veins to the heart. The right auricle is normal in size but unusually conspicuous in the frontal position (Figs. 4 and 5), suggesting that the heart has been rotated toward the

left as well as displaced; this interpretation was proven incorrect, however, by subsequent study in the left oblique and lateral positions in which the main axis of the heart was found to be rotated to the right. The right ventricle is not well visualized but appears to be normal; the pulmonic conus definitely is not enlarged, as shown in Figure 11. The pulmonary artery (Figs. 5 and 11), however, seems to be slightly enlarged and rotated toward the right side; the left main branch (Figs. 5 and 11) likewise is larger and lies more transversely in the chest, probably as the result of the rotation of the heart and pulmonary artery. The abundant vascularity of the left lung is strikingly contrasted with the few stunted vessels on the right side. The finding of decreased vascularity in the diseased lung is in harmony with the anatomical investigation of Wright (6), the combined anatomical and roentgenographic studies of Birkelo and Brosius (7), and the angiopneumographic studies of Lopo de Carvalho (8, 9).

Fig. 11.

Fig. 12.

Fig. 11. Contrast roentgenogram, lateral position. The inferior arrow indicates the right auricle, the posterior arrow the unfilled left auricle, and the anterior arrow the right ventricle. The pulmonic conus, the pulmonary artery and the left branch with its subdivisions are opaque. Note absence of visualization of right branches.

Fig. 12. Contrast roentgenogram, lateral position. The posterior arrow points to the left auricle now filled with opaque medium. The vertical arrow denotes the left ventricle. The ascending and descending positions of the aorta are well visualized. The transverse aorta is poorly defined.

The left auricle and ventricle are well visualized in Figure 9 and are normal in size and shape. The location of the left auricle directly behind the left ventricle in this position of the chest (left anterior oblique, 35 degrees) confirms the impression that the heart is rotated to the right, for normally this exposure is obtained only with a rotation of from 45 to 60 degrees. The widening of the aortic arch in the frontal position (Fig. 6) and its unusually transverse position in Figure 9 with a rotation of the thorax of 35 degrees show that the aorta, too, is rotated as well as displaced to the right. Except for displacement the branches from the aortic arch appear to be normal.

For the sake of completeness we shall review briefly a case of left-sided fibrothorax and bronchiectasis described recently (5) and which presents almost a mirror image of the changes exhibited by the present case. The heart and mediastinal structures were displaced toward the left instead of the right side and as a result

the left subclavian and innominate veins were kinked and foreshortened. Of greater interest, the heart, the pulmonary artery, and the aortic arch were rotated toward the involved left side so that these structures in the frontal view appeared as though in the right anterior oblique projection. No circulation to the involved lung could be detected, whereas the blood vessels to the uninvolved side appeared to be increased in size and number.

Our limited experience in fibrothorax consisting of these two cases does not warrant final conclusions regarding the cardiovascular changes in this condition. In both right- and left-sided involvement, the heart and mediastinum are shifted toward the involved side and in each case the rotation of the heart and great vessels was toward the diseased lung. Further study is necessary to determine the mechanism of this rotation and to find whether or not it is a constant occurrence. In both cases, there was striking diminution or absence of pulmonary blood vessels to the involved side.

SUMMARY AND CONCLUSIONS

In this report we have described a case of right-sided fibrothorax due to bronchiectasis. Attention was called to the inadequacy of the usual methods of study and to the value of contrast roentgenography of the heart and thoracic blood vessels in this condition. It was possible to identify the size, the shape, and the location of the superior vena cava, the cavities of the heart, the pulmonary circulation, and the thoracic aorta. The heart and the great blood vessels were displaced into the right side of the chest and also rotated in the same direction. There was no detectable enlargement or other abnormality of the heart. The left branch of the pulmonary artery was slightly enlarged and the pulmonary circulation to the left side increased, in striking contrast to the dwarfed right branch and the almost complete avascularity on the involved side. This report demonstrates the value of contrast roentgenography of the cardiovascular system in a patient having fibrothorax.

We wish to thank the Department of Roentgenology of Bellevue Hospital for co-operation in this study.

REFERENCES

(1) ROBB, GEORGE P., and STEINBERG, ISRAEL: A Practical Method of Visualization of the Chambers of the Heart, the Pulmonary Circulation, and the Great Blood Vessels in Man. Jour Clin. Investigation, **17**, 507, July, 1938.

(2) Idem: Visualization of the Chambers of the Heart, the Pulmonary Circulation, and the Great Blood Vessels in Man: A Practical Method. Am. Jour. Roentgenol. and Rad. Ther., **41**, 1–17, January 1939.

(3) Idem: Visualization of the Chambers of the Heart, the Pulmonary Artery, and the Great Blood Vessels in the Normal: A Preliminary Study (to be published).

(4) Idem: Visualization of the Chambers of the Heart, the Pulmonary Circulation, and the Great Vessels in Heart Disease: Preliminary Observations. Am. Jour. Roentgenol. and Rad. Ther., **42**, 14–37, July 1939.

(5) STEINBERG, ISRAEL, and ROBB, GEORGE P.: Mediastinal and Hilar Angiography in Pulmonary Disease: A Preliminary Report. Am. Rev. Tuberc., **38**, 557–569, November, 1938.

(6) WRIGHT, R. D.: The Blood Supply of Abnormal Tissues in the Lungs. Jour. Path. and Bact., **47**, 489–499, November, 1938.

(7) BIRKELO, C. C., and BROSIUS, W. L.: Roentgen Visualization of Pulmonary Arterial Circulation in Autopsy Material. RADIOLOGY, **31**, 261–292, September, 1938.

(8) LOPO DE CARVALHO: Les Aspects Radiologiques du Hile et Leur Interprétation. Lisboa Méd., **14**, 539, 1937, and Tenth Conference de l'Union Internationale contre la Tuberculose, p. 83, Lisbon, September 1937.

(9) LOPO DE CARVALHO and EGAS MONIZ: Visibility of the Pulmonary Vessels (Angiopneumography). Acta Radiol., **14**, 433–451, 1933.

PRIMARY ULCER OF THE JEJUNUM[1]

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WHILE ulcer of the jejunum secondary to a gastrojejunal anastomosis is not uncommon, primary ulcer of the jejunum in the absence of such operative interference is very rare.

Sestier (1), in 1829, described a case of acute peritonitis following spontaneous perforation of ulcer of the jejunum.

Wagner (2) described the case of a young man, 18 years of age, who died as a result of a wall collapsing on him. At autopsy, at a point six inches distal to the duodenojejunal junction and opposite the mesentery, there was a sharply delimited oval perforation; the margin was partly undermined. The lesion had the appearance of a chronic ulcer of the jejunum which had temporarily been prevented from perforation by adhesions to the great omentum. As a result of the accident, the adhesions were broken loose with ensuing perforation and peritonitis. Wagner believed that the ulcer was the result of the corrosive action of gastric juice although in an attenuated degree.

Taylor (3), in 1865, described the case of a man, aged 45 years, who after violent physical exertion complained of intense abdominal pain and died. At autopsy a severe peritonitis was found, and a perforation of the jejunum the size of the head of a pin. The opening appeared to have been the result of an ulcer which gave way as the result of abdominal strain.

Reverdin's (4) patient was a male, 55 years of age, with a vague history of abdominal trauma. Autopsy revealed a punched-out ulcer situated on the free border of the intestine in the mid-portion of the jejunum.

In Brigidi's (5) case of ulcer of the small intestine, the condition was found in a male, aged 40 years, who died of pulmonary disease. Autopsy revealed ulcers in the jejunum and ileum, the specimen being preserved at the Pathological Institute, at the University of Genoa. Pathological examination revealed no evidence of caseation or gummas. There was no evidence of nephritis. It was his opinion that the ulcers arose on the basis of catarrhal inflammation.

Simpson (6) described the case of a man, 56 years old, who at the onset of his illness complained of slight pain in the region of the stomach and vomiting. By the following morning he was found in a state of collapse and died shortly thereafter. Autopsy revealed a single ulcer of the jejunum, about six inches distal to the duodenojejunal junction. The ulcer was six millimeters in diameter, irregularly circular in outline, with clean-cut edges, and had perforated through all the coats. There was no evidence of chronicity. The ulcer had an appearance similar to that of a perforating gastric or duodenal ulcer. Simpson considered the possibility that the jejunal ulcer might have originated as the result of trauma from a foreign body, although no such substance was found in the alimentary canal, except a few shreds of partially masticated wood fiber in the colon.

In the case described by Dodson (7), perforation of a small chronic ulcer of the small intestine about eight feet from the stomach occurred in a 57-year-old male. The perforation followed abdominal trauma.

Jankowski (8) described a case of perforation of a jejunal ulcer in a male, aged 48 years. Autopsy revealed a funnel-shaped perforated ulcer the size of a pea, which he considered to be a simple ulcer. Near the perforated lesion was a second jejunal ulcer which had not perforated.

Schmilinsky (9), in 1910, reported the case of a woman, 63 years of age, who had complained of abdominal pain for years and who had suffered a hemorrhage from the

[1] This is one of a series of papers contributed by friends and former pupils of I. Seth Hirsch, M.D.

alimentary tract on two occasions. Operation showed a stenosing ulcer of the jejunum four centimeters distal to the duodenojejunal junction. Pathologically the ulcer resembled a gastric or duodenal ulcer.

Arkin (10) described the pathologic findings in a male, 65 years old. A history could not be obtained as the patient was in a coma when admitted. Autopsy revealed, in addition to a gummatous meningitis, gummas of the liver, spleen, and kidney, a syphilitic osteitis, and the presence of several ulcers of the jejunum. One ulcer was also found at the splenic flexure and another of a similar nature in the rectum. A detailed study of one of the jejunal ulcers was made. Arkin believed them to be of syphilitic origin and described the presence of the spirochaeta pallida in the lesion.

In the report by Cade, Roubier, and Martin (11), a perforation of a jejunal ulcer occurred 60 centimeters from the duodenum in a male, aged 25 years. Microscopic study of the margin of the perforated ulcer showed an intense inflammatory infiltration. There was no evidence of any specific infection. Pathologically it had the appearance of a simplex ulcer.

In Richardson's (12) case, the patient, 47 years old, gave a history of recurring attacks of severe general abdominal pain. Following one attack he noticed black stools. Roentgen examination was negative for renal calculi; no gastro-intestinal roentgen examination was made. At operation an indurated area was felt in the small intestine, due to a stricture, on the anterior surface of which was a perforation one-eighth inch in diameter. The intestine was opened, and an annular ulcer was found encircling the bowel at the point of the stricture. This appeared to be about two feet distal to the duodenum. The ulcer was resected. Microscopic examination showed evidence of a chronic ulcer.

The records of the Massachusetts General Hospital showed only one similar case; this was in a 48-year-old female. Operation revealed a perforation of an ulcer, apparently in the jejunum. Microscopic examination showed it to be a simple ulcer. There was no evidence of either syphilis or tuberculosis in either of the two ulcers reported.

Barber (13), in reporting his case of primary ulcer of the jejunum, stated that in a personal communication Dr. Symmers, Director of the Pathological Laboratory at Bellevue Hospital, had stated that he had no record of any preceding case. Barber's patient had been operated on twice for a perforated gastric ulcer. Finally, after being symptom-free for some time, he had a sudden attack of severe pain in the region of the umbilicus which necessitated a third operation. This time, in addition to a generalized peritonitis and a healed ulcer of the pylorus, there was a perforated and moderately indurated ulcer of the jejunum one centimeter in diameter, and about two centimeters distal to the duodenojejunal junction. The patient made an excellent post-operative recovery.

Ebeling (14) found two cases of primary ulcer of the jejunum at the University of Pennsylvania Hospital between the years 1921 and 1932. Including his own personal case, he found a total of 47 cases described in the literature from 1927 to 1932. A radiographic study made in his case showed stasis in the duodenum and jejunum with obstruction in the proximal jejunum. The roentgen diagnosis was ulcer or adhesions. The clinical preoperative diagnosis considering the clinical history was jejunal ulcer. Operation revealed a stenosing lesion of the jejunum, six inches from the ligament of Treitz. Approximately four inches of jejunum were resected. Pathologic examination disclosed a shallow ulcer.

According to Ebeling, the majority of the ulcers were located in the upper jejunum. Of those that had not perforated, four were in the first loops of jejunum, two were in the upper jejunum, and one in the mid-jejunum. Of those that had perforated, nine were in the upper loop, ten in the upper jejunum, four were in the mid-jejunum, and four in the lower jejunum. The majority of these ulcers were localized opposite the attachment of the mesentery.

Stricture of the bowel at the site of ulceration was commonly present. In Ebeling's case, the stenosis was due mainly to a constricting spasm of the ulcer-bearing area. The ulcers were described as being punched-out, predominantly circular, and similar in appearance to chronic ulcers of the stomach and duodenum. In size, they varied from about four millimeters to two centimeters. In those ulcers that had perforated, the site of perforation was often minute. Microscopic examination showed round-cell infiltration and fibrosis. In the few cases in which enlarged mesenteric lymph glands had been reported, examination showed changes due to chronic inflammation. In nine of the cases, additional ulcers were found.

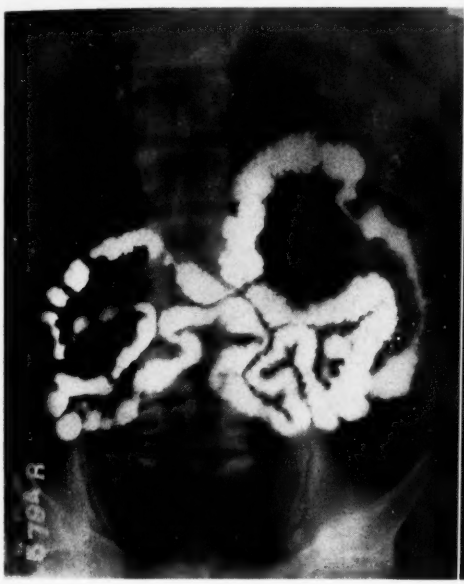

Fig. 1.

Fig. 2.

Fig. 1. Appearance of narrowed jejunum 45 minutes after the ingestion of barium.
Fig. 2. Appearance of narrowed jejunum one hour after the ingestion of barium.

There is no record of any case of primary jejunal ulcer showing the presence on histological examination of any aberrant tissue either gastric or pancreatic.

In addition to the case of primary jejunal ulcer which was radiographed by Ebeling, further reports of radiographic examinations in such cases are those of Murphy, Walton, Ravdin, and Harris.

In the case reported by Murphy (15), radiographic examination revealed obstruction to the outlet of the stomach and involvement of the gall bladder. The preceding history had been that of gastric disturbance for 15 years. The attacks consisted of epigastric pain occurring from one to three hours after meals. The pain was relieved by vomiting but not by food. There were tarry stools on one occasion. The pre-operative diagnosis was duodenal ulcer. At operation, an ulcer of the duodenum was found. In addition, another ulcer was found in the first portion of the jejunum associated with slight stenosis. Murphy stated that this was the second time he had seen a primary ulcer of the jejunum. A posterior gastro-enterostomy was done. The ulcer remained *in situ*, so that no histologic study was possible.

Walton (16) reported the case of a woman,

45 years old, with a history of recurring attacks of "stomach trouble" for 19 years. The pain, located in the epigastrium, occurred several hours after eating and was often relieved by food. Occasionally the pain woke her from sleep about 2 A.M. The symptoms gradually became worse. Roentgen examination failed to disclose any organic lesion, and operation revealed the stomach, duodenum, gall bladder, and appendix to be normal. However, in the jejunum, three feet from the duodenojejunal flexure, there was an area of induration, two inches long, to which the omentum was adherent. The area of involvement was resected. On opening the intestine, a round ulcer similar in appearance to that of a gastric ulcer was seen. Histologic examination disclosed only evidence of chronic inflammatory changes.

In Ravdin's (17) case, the patient complained of pain in the epigastrium and right upper quadrant, which radiated across the upper abdomen to the left and occasionally to the back. The pain occurred about two hours after meals, and was not relieved by food. Radiography showed obstruction high up in the jejunum. At operation, there was an annular sclerosing mass encircling the jejunum about twelve inches from the duodenaljejunal junction. The mass was resected, the diagnosis being sarcoma or carcinoma. Pathological examination of the margins showed chronic inflammatory tissue. The final diagnosis was chronic lymphadenitis and chronic jejunal ulcer.

Owing to the neutralization of any excess gastric acid, Ravdin believed it highly improbable that peptic digestion plays any etiologic rôle.

In the case reported by Harris (18), the patient, a male aged 48 years, had complained of upper abdominal pain and vomiting of bile-stained material. After subsidence of the attack, symptoms recurred suggestive of a gastric ulcer. Radiographic examination showed a niche in the left upper quadrant which was interpreted as being due to an ulcer on the posterior wall of the stomach. Autopsy revealed two perforated jejunal ulcers opposite each other—one on the anterior and one on the posterior wall. The location of the ulcers was four inches beyond the duodenojejunal junction. The benign nature of the lesions was corroborated by microscopic study. Nothing is stated in the report regarding the presence of aberrant gastric or pancreatic tissue. The niche originally noted in the radiographic examination and which had been interpreted as being due to a gastric ulcer had evidently resulted from barium retention in a jejunal ulcer.

Recently we have had a personal experience with a case of primary jejunal ulcer which was carefully examined radiographically, and the diagnosis of jejunal ulcer established at operation.

This patient, V. C., aged 52, was accustomed to going on drinking bouts during which he took hard liquor steadily for from eight to twelve hours, and this he repeated every few weeks. Three weeks before admission he went on an alcoholic spree. Shortly thereafter he had griping upper mid-abdominal pain and vomiting. He felt better after the vomiting, but continued to have sharp, agonizing pain localized mid-way between the xyphoid process and the umbilicus. These pains were severe enough to cause him to double up at times. The pain kept him awake at night.

A radiographic study of the stomach and duodenum, as well as of the small intestine was made. This special study of the small intestine consists of the following procedure: After the ingestion of the barium, several films are taken of the stomach and duodenum; a 14 × 17 film is then taken every 15 minutes during the period of one hour. This is followed by a similar examination at two, three, five, six, and nine hours.

Radiographic examination of the proximal jejunum revealed an area of about one and one-half inches in length that was markedly narrowed. The contour was fairly smooth in outline. Along the lateral border of the narrowed area, at its proximal

region was a persistent small niche-like projection (Figs. 1, 2, and 3).

Diagnosis.—The roentgen diagnosis was inflammatory jejunitis. A newgrowth could not be entirely ruled out. The significance of the niche-like area in the involved jejunum, persistent in all the films, was not recognized. About four days after the completion of the radiographic study, the patient developed the clinical evidence of an acute abdomen which necessitated immediate surgical intervention. As determined at operation (performed by Dr. Berry) and as noted in the resected specimen, this niche-like area presumably represented the region through which perforation occurred.

Fig. 3. Appearance of narrowed jejunum two hours after the ingestion of barium.

Operation revealed the following: In the mid-portion of the jejunum was a small perforation on its mesenteric border. Proximal to this, the intestine was considerably dilated; distal to the perforation it was contracted. The peritoneal cavity beneath the gastric omentum was filled with turbid fluid and with occasional flecks of fibrin. The diseased area of jejunum was resected.

Macroscopic Examination.—The specimen consisted of a piece of the jejunum 20 cm. in length. The proximal 12 cm. was dilated. The intestinal wall here was thicker than normal. The folds of the mucosa were flattened and the subserosal blood vessels somewhat dilated. There was a constriction of the intestinal wall 12.5 cm. from the proximal end. Here the wall was slightly thickened for 2 cm. and the mucosa flattened. There was an opening in the mucosa on the mesenteric border 5 mm. in diameter about which the mucosa

Fig. 4. Reproduction of resected specimen of jejunum showing perforated ulcer with a probe through it.

was thickened and harder than normal. The distal 7.5 cm. of the jejunum was normal in diameter, the serosal and mucosal surfaces appearing natural. The probe passed through the opening for 8 mm. toward the mesentery proximally and perforated the serosa. On section, the tract was brown, the wall of the intestine thickened and grayish white in color.

Microscopic Examination.—The mucosa showed edema, increased vascularity and congestion of the blood vessels, and infiltration with plasma cells, lymphocytes, and numerous eosinophiles. The muscularis was slightly thickened and a few chronic inflammatory cells were present. The serosa was thickened; congested blood vessels were present; inflammatory cells and an occasional giant cell were seen. Large giant cells were seen among the fat cells of the mesentery. Sections through the tract show that the latter was lined by chronic inflammatory tissue. The tract penetrated through the entire intestinal wall into the mesentery where the acute inflammatory reaction was intense. Numerous blood vessels in the mesentery showed thickening of the media (Fig. 4).

Comment.—This case is of interest for the following reasons:

1. The extreme rarity of primary jejunal ulcer.

2. The value of the fractional method of studying the small intestine as outlined above in the roentgen demonstration of organic lesions of the small bowel.

3. It is worthy of note that pathological examination revealed no evidence of any aberrant tissue, either gastric or pancreatic. In none of the cases previously reported of primary jejunal ulcer, has there ever been any evidence of aberrant gastric or pancreatic tissue.

4. The rôle of gastric acidity in the genesis of primary jejunal ulcer cannot be considered as having any significance because of the absence of aberrant gastric tissue, and because it is difficult to assume that gastric secretion leaving the stomach and duodenum unaffected would retain sufficient power to continue as a factor in the development of an ulcer in the jejunum well beyond the ligament of Treitz.

REFERENCES

(1) SESTIER: Perforation du Jejunum ayant cause une Peritonite Aigue. Bull. de la Soc. Anat. de Paris, **4,** 33, 1829.

(2) WAGNER, E.: Fall von perforirendem Geschwür in Anfange des Jejunum, Schlag auf die untere Korperhälfter, Rupture der Niere, Peritonitis. Tod. Archiv. f. Physiolog. Heilkunde (new series), pp. 280–283, 1858.

(3) TAYLOR: Death from Perforation of the Jejunum as a Result of Disease: Death Apparently a Result of Violence. Guy's Hosp. Rep., **11,** 282, 1865.

(4) REVERDIN: Perforation du Jejunum. Peritonite subaigue. Mort: autopsie. Bull. de la Soc. Anat. de Paris, **7,** 597, 1867.

(5) BRIGIDI: Larghe Ulcere croniche nel tenue intestino. Gazz. d. osp., **14,** 1050, 1893.

(6) SIMPSON, F. O.: Acute Perforating Ulcer of the Jejunum. Practitioner, **58,** 165, 1897.

(7) DODSON: Death from Rupture of Intestinal Ulcer. British Med. Jour., **1,** 718, 1906.

(8) JANKOWSKI, J.: Perforative Peritonitis bei einem Ulcus simplex des Jejunum. Deutsche med. Wchnschr., **34,** 2267, 2268, 1908.

(9) SCHMILINSKY: Ein Fall von Stenosierendem Ulcus chronicum des obersten Jejunums. München. med. Wchnschr., **57,** 1616, 1910.

(10) ARKIN: Acquired Syphilitic Ulcers of the Bowel. Tr. Chicago Path. Soc., **8,** 224, 1909–1912.

(11) CADE, A., ROUBIER, C. H., and MARTIN, J. F.: Les ulceres simples du jejuno-ileon. Progrès méd., **29,** 309–312, 1913.

(12) RICHARDSON, E. P.: Jejunal Ulcer without Previous Gastro-enterostomy. Surg., Gynec. and Obst., **35,** 1–10, July, 1922.

(13) BARBER, W. H.: Ulcer of the Jejunum. Ann. Surg., **74,** 621, 1926.

(14) EBELING, W. W.: Primary Jejunal Ulcer. Ann. Surg., **97,** 857–874, June, 1933.

(15) MURPHY, J. B.: X-ray Findings in Primary Jejunal Ulcer. Surg. Clin. Chicago, **5,** 435, 1916.

(16) WALTON, A. J.: Primary Jejunal Ulcer. British Jour. Surg., **10,** 152, 153, July, 1922.

(17) RAVDIN, I. S.: Primary Ulcer of the Jejunum. Ann. Surg., **75,** 873–878, 1927.

(18) HARRIS, JOHN H.: Primary Jejunal Ulcer: A Case Report. RADIOLOGY, **26,** 497, 498, April, 1936.

BONE RAREFACTION AFTER TRAUMA TO LARGE JOINT REGIONS WITHOUT FRACTURE[1]

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LOCAL regressive soft-tissue changes after an acute trauma without fracture to a large joint region are fairly common and familiar. For instance, it is recognized that a violent wrench, blow, or bump to the knee can lead to severe and disabling local muscle atrophy. Occasionally, the latter is even associated with the presence of one or more small areas of rarefaction in the spongiosa near the joint. Very rarely, without having induced a fracture, an acute trauma to a large joint region instigates, in addition to soft-tissue changes, an impressive degree of bone rarefaction. In these cases, the rarefaction extends far along one or all of the bones entering into the formation of the joint, and involves both the spongiosa and the cortex. That this sequence of events can occur at all does not seem to be well known.

Fig. 1. Fig. 2.

Fig. 1. Roentgenograph showing extensive rarefaction of femur; note severity of changes in condylar region.

Fig. 2. Roentgenograph showing the rarefied femur of Figure 1 in the anteroposterior projection.

Cases presenting rarefaction of long bones after trauma (without fracture) to large joint regions are analogous in many respects to cases showing rarefactions of hand or foot bones after trauma (not infrequently without fracture) to the wrist or ankle region. Though fairly familiar clinically and roentgenographically, the latter such cases, often discussed under the head of "Sudeck's atrophy" or "Leriche's dis-

[1] This is one of a series of papers contributed by friends and former pupils of I. Seth Hirsch, M.D.

ease" of the hand or foot, are but poorly understood anatomically.[2] Indeed, there has been practically no opportunity for anatomic examination of adequate material from cases of post-traumatic rarefaction of the hand or foot bones. As a matter of fact, there seems to be no satisfactory discussion of the pathology of post-traumatic (but not post-fractural) rarefaction of bones in any location. In the present paper, an attempt is made to supply this deficiency, at least in part, and to correlate the pathologic with the roentgenographic findings in such cases. A discussion of the pathology of post-traumatic rarefaction of long bones ought to shed some light upon post-traumatic rarefaction of the hand or foot bones as well. It should likewise be of help in understanding the changes which take place during the pre-collapse stage in post-traumatic rarefaction of vertebral bodies (Kümmell's disease).

Clinical and Roentgenographic Aspects.—As has already been pointed out, cases of pronounced bone rarefaction following upon trauma (without fracture) to the region of a large joint are extremely rare. In these cases, the overlying soft parts also become modified in various ways. In particular, there is usually some muscle atrophy, and the skin may present changes attributable to local vasomotor and trophic disturbances. The region may be slightly swollen or perhaps only appear so on account of thinning of the limb above and below it in consequence of muscle atrophy.

Sinding-Larsen (4) has described an instance of pronounced post-traumatic rarefaction in which it was the knee joint region that was affected. The case was that of a man, 40 years of age, who was struck on the inside of the right knee by a falling herring barrel. This knee swelled immediately and gradually became stiff. For 11 months subsequently, the patient was treated on the assumption that the condition was a low-grade osteo-articular tuberculosis. Then, still suffering from pain, swelling, and limitation of motion in the knee, he came under the care of Sinding-Larsen, who also thought at this time that the condition was a tuberculous gonitis. However, the latter disease was excluded by a negative tuberculin reaction and by the peculiar "spotty" rarefaction detectable roentgenographically in the femur, patella, and tibia. Diathermy and cautious exercise of the part brought about prompt clinical recovery and incidentally showed that the condition was not tuberculosis. However, it is significant that even 27 months after the injury, rarefied spots were still discernible roentgenographically, though with difficulty, in the affected knee-joint bones. Since there was no operative intervention, no opportunity was afforded for anatomic study of this case.

Roentgenographs from another case of post-traumatic rarefaction of the knee are pictured in Haglund's text on orthopedics (5). No specific anatomic data are given on this case. Recently, Sudeck (6) too has discussed an instance of post-traumatic rarefaction of the knee region. His subject, a man 52 years of age, had received a relatively slight injury to the knee, which was painful for only a day or so at first. After ten days he again began to suffer from pain, and motion became limited in

[2] In this connection, it seems appropriate to consider briefly the question of so-called "acute bone atrophy." Indeed, from the roentgenographic point of view, post-traumatic rarefaction of hand or foot bones belongs in the category of "acute bone atrophy." The initial modification is represented by an unclear, hazy appearance, especially of the cancellous ends of the metacarpal or metatarsal bones and of the carpal or tarsal bones. From about two to three months after the trauma, these regions, and also the spongy ends of other affected bones, often show small, roundish, closely set rarefactions. The affected bones now present a distinctly "mottled" or "spotted" appearance which is characteristic of the early stage of "acute bone atrophy." It should be pointed out, however, that a "mottled" or "spotted" appearance is observed roentgenographically in the hand and foot bones in a variety of other conditions also. In fact, such pictures were first described by Sudeck himself (1) in connection with inflammations of interphalangeal joints and soft tissues of the hand or foot. Dubs (2) observed spotty bone atrophy as a sequel of severe burns of the hand or foot; it also appears after freezing of these parts. The present writer has seen it in the hand of a patient suffering from neuritis and herpes zoster. Jaffe and Pomeranz (3) described it as occurring in the foot bones in connection with vascular disease of the lower limbs.

the affected region. Three months after the injury, the bone rarefaction was already clearly apparent roentgenographically. The case was also interesting in that it was a source of medico-legal controversy. That it became such is not surprising in view of the slightness of the original injury. No pathologic material became available for study in this case either.

The present writer has had the opportunity of studying material from a case showing a particularly severe post-traumatic rarefaction of a knee region.[3] In this case, the rarefaction was so conspicuous, especially in the femur, that a malignant tumor was thought to have developed in this bone and consequently the limb was amputated through the upper third of the thigh. This erroneous diagnosis, with its baleful consequences, is understandable in view of the deceptive roentgenographic appearances. The subject, a man 30 years of age, had jumped from a wagon and injured his right knee; he was unable to continue his work that day. Subsequently, he resumed his work, but accomplished it with some difficulty because of pain and limitation of motion of the knee. Nine weeks after the injury, there was some flexion deformity of the region and enough soft-tissue swelling to obliterate the bony points, and the knee region was tender to touch. Otherwise, the general health of the patient was good, and the body temperature was not increased. Clinical laboratory examinations showed nothing abnormal.

Roentgenograms, taken about ten weeks after the injury, show the knee in a position indicating flexion deformity. The lower half of the femur presents profound and extensive modification (Fig. 1). Specifically, the condylar regions reveal complete obliteration of their spongy architectural pattern. Instead of this, the lower end of the femur presents an obfuscated, cloudy, somewhat mottled ground-glass appearance. Furthermore, the condylar outlines show notches in some places and in others are so vague that they

Fig. 3. Roentgenograph showing post-traumatic rarefaction of the lower end of the humerus.

can hardly be traced. Proximally to the condyles, the rarefaction is still very pronounced. Viewed laterally, the shaft cortex shows longitudinal tracts of rarefaction, some of which are quite near its surface. In the anteroposterior projection, the extensiveness of these areas becomes apparent (Fig. 2). Nowhere along the femur is there any evidence of deposition of new bone by the periosteum. The patella, too, presents mottled rarefaction, and irregularity and obscuration of its outline. In general, the tibia and fibula for some distance below their upper ends are also so modified. In these bones, however, the alterations are by no means as pronounced as in the femur. The interpretation of these roentgenographic changes on the basis of the pathologic findings will be given presently.

The writer has, in addition, studied a case of post-traumatic rarefaction of the elbow region (Fig. 3). In this case, the changes were by no means so pronounced

[3] I am greatly indebted to William Boyd, M.D., now Professor of Pathology at the University of Toronto, for tissue and copies of roentgenographs, sent me some years ago, from this very unusual case, and for his kind permission to use this material.

as in the one just described. The subject was a young adult who had received a relatively mild injury to the outer side of the elbow which was followed by pain and considerable disability. A roentgenograph taken some time later disclosed the presence of rarefaction, involving the lateral epicondyle and the adjacent portion of the humeral shaft. The outline of the affected part of the humerus was irregular, and, in the anteroposterior projection, large roundish areas of rarefaction were also visible. In this case, too, material became available for anatomic study. A biopsy was performed, and the diagnosis of post-traumatic rarefaction was established.

Fig. 4. Roentgenograph of transverse slice from femur shown in Figure 1; the slice was taken about three inches above the knee.

Pathology.—Professor Boyd's note, accompanying the material from his case, gives some information on the gross changes in the femur. It appears that the outer surface of the lower end of this bone was found superficially undermined in some places while in others it had actually been eroded. In the interior of this part of the femur there was extensive resorption of the spongy osseous tissue, the latter being represented only by scattered gritty fragments embedded in fluid fatty marrow. The articular cartilage of the femoral condyles was not found altered.

The writer received for study a block of shaft and some modified spongiosa. A roentgenogram of a transverse slice of this block shows quite well the superficial undermining of the outer cortical surface and the complete erosion of this surface in some places (Fig. 4). It also shows a very uneven mottled rarefaction within the cortex. In certain of the more conspicuous rarefied areas, numerous thin-walled blood vessels embedded in a loose, fatty connective tissue were detectable, even grossly.

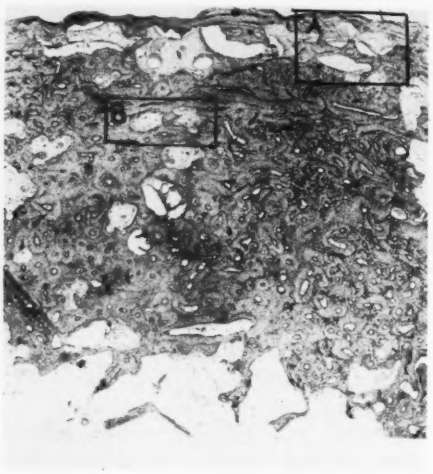

Fig. 5. Photomicrograph (low-power survey) showing the distribution of the rarefaction throughout the thickness of the cortex.

Sections made from transverse cortical slivers embedded in celloidin were examined microscopically. In these it can be seen that the diameters of a large proportion of the haversian canals are increased. Furthermore, the outlines of many of these canals are distorted, and the blood vessels which they contain are dilated and often engorged. One can also see an abnormal number of perforating (Volkmann) canals radiating from the enlarged haversian canals. Here and there, but particularly near the cortical surface, groups of enlarging canals have coalesced through resorption of the intervening osseous tissue, to form the large spaces visible grossly and appearing roentgenographically as rarefactions (Fig. 5). These spaces contain a loose and edematous

fibro-fatty marrow carrying numerous large, and, in life apparently engorged, thin-walled blood channels. Some of these channels abut directly upon the osseous borders of the spaces. The outlines of the spaces are irregular, and, even in connection with any one space, part of the wall may show osteoclasts in Howship's lacunæ (evidence of resorption) while another part of the wall may be found already lined by osteoblasts and smoothed out by newly deposited bone (Figs. 6 and 7).

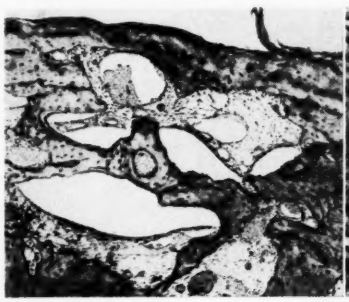

Fig. 6.

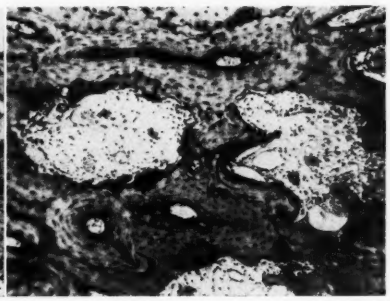

Fig. 7.

Fig. 6. Photomicrograph (moderate magnification) of area blocked out and marked *A* in Figure 5, showing the character of the enlarged and coalesced vessel spaces immediately under the cortical surface.

Fig. 7. Photomicrograph (moderate magnification) of area blocked out and marked *B* in Figure 5, showing the resorption and new bone deposition on the walls of the large cortical spaces.

In addition to enlargement of haversian canals and the formation of spaces representing their coalescence, there are also changes in the osseous tissue about and between the canals (Fig. 8). In the first place, the proportion of interstitially located bone has increased at the expense of the bone arranged around the canals to form haversian systems. Furthermore, the cement lines separating the haversian systems from the interstitial bone are more numerous, thicker, and much more irregular than they would normally be. These various changes are additional indications of the lively reconstruction which has been going on in the cortex of this femur. In regard to the interstitial osseous tissue, it is also to be noted that nuclei are largely absent from its bone-cell lacunæ. This suggests that this tissue is partially necrotic, although in interpreting this necrosis it should be remembered that nuclei also tend to be lacking in the interstitial osseous tissue of normal adult femoral cortex (3). However, the osseous tissue about the haversian canals (except in instances in which there is evidence that the haversian systems are undergoing reconstruction) does show bone cells in the bone-cell lacunæ almost throughout.

What is interesting when one examines the periosteal surface of the cortex is the absence of osteophyte-like new bone deposition by the periosteum. Not only has there been no re-enforcement of the cortex, but, instead, some of the large cortical spaces have extended until they are directly beneath the periosteum, producing defects in the cortical surface. On the medullary side of the cortex, abnormal spongification of the latter has occurred in some places. In the spongiosa proper, the intertrabecular marrow is fatty, somewhat abnormally fibrillar, and slightly edematous (Fig. 9). It contains, in general, a meager sprinkling of cells, mainly mononuclears, and here and there a small agglomeration of them. The original osseous trabeculæ have been largely resorbed, but such trabecular fragments as have persisted show evidence of reconstruction and regeneration.

Pathogenesis.—We do not know the instigating and mediating mechanisms responsible for the fact that trauma to a joint region is often followed by muscle atrophy and other local soft-tissue changes, and, sometimes, in addition, by pronounced bone rarefaction. It is probable that whatever these mechanisms may be, they are the same for the soft tissue as for the bone changes. The answer to this problem of pathogenesis seems to lie in the realm of neurophysiology, and more specifically in the field of neurovascular dynamics. However, mere disuse, even with the implication that it results in diminished nutrition, can hardly explain the whole complex of soft-tissue and bone changes in the affected part. On the contrary, the condition may develop despite continued use of the traumatized region, and furthermore, the anatomic changes in the femur described previously are not those characteristic of the simple smooth atrophy of inactivity. Notably, one does not see, in bones undergoing atrophy caused by inactivity, the numerous dilated and engorged blood vessels which are so prominent in the condition under consideration here. This condition of the vessels is certainly an important factor in the enlargement and erosion of the haversian canals and in the general rarefaction of the bone. It is possible that the state of dilatation of these vessels is due to a sort of reflex paralysis of their walls. This idea is suggested by the fact that in the cases of post-traumatic rarefaction of hand or foot bones the overlying soft parts are livid and cold.

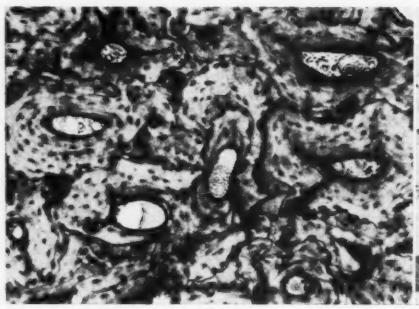

Fig. 8.

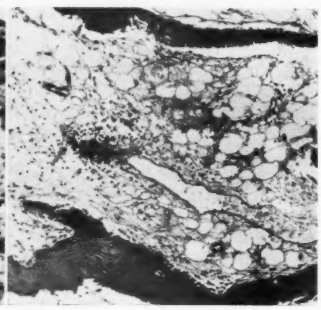

Fig. 9.

Fig. 8. Photomicrograph (moderate magnification) showing a more compact portion of the cortex; there is distortion of the outlines of the haversian systems and an excessive amount of interstitial bone as evidence of lively reconstruction.

Fig. 9. Photomicrograph (moderate magnification) showing spongy trabeculæ in the process of resorption and reconstruction; the edematous intertrabecular fatty marrow contains a collection of mononuclears.

DISCUSSION AND CONCLUSIONS

The preceding exposition has shown that an acute trauma (without fracture) to a large joint region may instigate severe rarefaction in the long bones of the vicinity. In association with this rarefaction, there are regressive changes in the overlying soft tissues. In particular, the muscles are likely to become very atrophic. The post-traumatic rarefaction in question is definitely uncommon. It has been noted more often in the knee region than elsewhere. Because it is rare and because the possibility of its occurrence is but little appreciated, the condition is usually misdiagnosed, at least when first encountered. Specifically, of the three instances cited in detail in which the knee region was involved, the first was held to represent a tuberculous arthritis, the second some other infectious arthritis, and the third a malignant bone tumor.

It is not surprising that such errors should occur. In fact, they are often made even in connection with post-traumatic

rarefaction of the hand or foot bones, which is much more common. Subjects affected with this condition are sometimes mistakenly held to be suffering from tuberculosis of the wrist or ankle bones. Or, instead, they are even labeled as neurotic because of the severity of the pain complained of in comparison with the objective changes. In cases in which this error is committed, the bone rarefaction is usually ascribed to mere inactivity. Again, the writer has seen an instance of traumatic rarefaction of the foot bones in which the subject was supposed to have a malignant tumor of the os calcis.

The instigating trauma in cases of post-traumatic rarefaction is sometimes a slight one, and may hardly be remembered by the patient. Already on this account, the condition may be incorrectly evaluated in some instances. That it can be misinterpreted even when it is recognized as being connected with a previous trauma has heretofore been indicated. It becomes apparent that in certain cases medico-legal injustices may result from such errors. On the other hand, a history of trauma is so common in skeletal disorders in general, that one should beware of concluding too readily in a given case that one is dealing with a post-traumatic rarefaction.

Anatomically, the bone rarefaction is manifested in porousness of the compacta and meagerness of the trabeculæ of the spongiosa. The rarefaction can be seen to be dependent upon hypervascularization, which is particularly prominent in the cortical bone. In the latter, the large resorption spaces are found filled with a loose, fibro-fatty connective tissue bearing numerous engorged blood vessels. If the bone regions rarefied are such as normally have thin cortices, these regions are likely to become so greatly weakened as to yield easily under functional strain. It may be on this account that persons with post-traumatic rarefaction of the hand or foot bones suffer so much pain when the affected parts are used. It may likewise be for this reason that vertebral bodies sometimes collapse after trauma, coming to manifest Kümmell's disease.

Post-traumatic rarefaction is a stubborn condition. Indeed, it is but slowly, if at all, that the affected bones re-acquire a completely normal roentgenographic appearance. However, even before there is substantial regression of the rarefaction, considerable articular function is usually already possible. Recovery is definitely favored by active use of the affected part, in association with physiotherapeutic measures such as diathermy and massage.

BIBLIOGRAPHY

(1) Sudeck, P.: Arch. f. klin. Chir., **62,** 147, 1900.
(2) Dubs, J.: München. med. Wchnschr., **68,** 1141, 1921.
(3) Jaffe, H. L., and Pomeranz, M. M.: Changes in Bones of the Extremities Amputated Because of Arteriovascular Disease. Arch. Surg., **29,** 566–588, October, 1934.
(4) Sinding-Larsen, C. M. F.: Acta Radiol., **3,** 515, 1924.
(5) Haglund, Patrik: Prinzipen der Orthopädie (see p. 69), G. Fischer, Jena, 1923.
(6) Sudeck, P.: Hefte zur Unfallheilkunde. F. C. W. Vogel, Berlin, 1938.

THE TANGENTIAL IRRADIATION OF BREAST CARCINOMA[1]

By N. S. FINZI, M.B. (Lond.), D.M.R.E. (Camb.), *London, England*

DURING a visit to the United States in 1937, I realized that the tangential method of treating malignant disease of the breast was by no means as widely used as it was in England. I feel, therefore, that Dr. Hirsch, though he knows the method well and employs it himself, will welcome a few words from me on this subject.

Fig. 1.

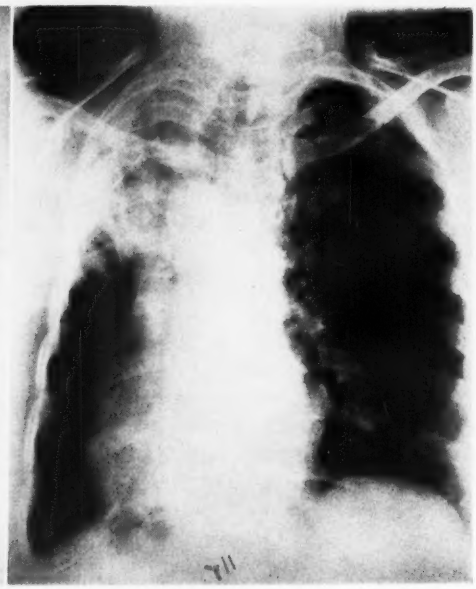

Fig. 2.

Fig. 1. Patient treated by three overlapping fields. Skiagram made one year after the commencement of treatment shows mottling in the lung, drawing across of the aorta, and drawing up of the left diaphragm. Before the treatment the chest was normal.

Fig. 2. Same patient ten years later. Note the more normal position of the diaphragm and the bronchiectatic cavities toward the apex.

Until the latter part of 1921, I treated cases of breast carcinoma by three overlapping fields, at right-angles to the surface, giving the treatment at intervals of three weeks for about six months and often a second course of treatments for a period of three months. For years the voltage and filtration were gradually increased. Probably on account of this I began to realize, about the beginning of 1921, that some of the cases which we thought were recurrences in the lung, were, in fact, damage following x-ray treatment. These occurred only in some instances and were definitely more likely to occur when there had been old healed lung disease. As I had made x-ray examinations of the chest and mediastinum a routine in breast cases, this fact was soon established, and it seemed highly probable that the effect was due to a bacterial invasion of damaged tissues. It took some time to convince my radiological colleagues that this was true,

[1] This is one of a series of papers contributed by friends and former pupils of I. Seth Hirsch, M.D.

but eventually the evidence became too strong to be controvertible.

Something had to be done, so, in the middle of 1921, I instituted the tangential or glancing method. Soon after this, I increased the filtration. A little later, I ordered the glancing method for a patient, but, owing to a misunderstanding, she was treated by the old method with the new filtration. The result was an acute burn of the lung, showing first about three weeks later. The case was followed through for years. The characteristics are, in the acute stage, a faint shadow like a pneumonia, but with less pain and no fever. This stage can only rarely be seen, but the later stages are the same in the acute and chronic cases. The most characteristic feature of the later stage is an extreme displacement of all the mediastinal contents toward the affected side (Figs. 1 and 2). Later they gradually return toward the normal position but never reach it: as they do this, a characteristic mottling is seen in the affected lung with clear spaces of an initiate bronchiectasis (Fig. 3). After several years, the appearance becomes that of typical bronchiectasis, though very often symptomless. In some cases the symptoms have been those of a chronic bronchiectasis: more often there is a tendency to bad bronchitis in the winter which eventually passes off. The mechanics of the condition are probably as follows: first, a sticky secretion in the smaller bronchi which blocks these bronchi, causing collapse of the lung; the mediastinal contents are, therefore, dragged across to the affected side as is usual with a blocked bronchus; bronchiectasis then supervenes and partially clears up later, if the blockage of some of the bronchi disappears.

The introduction of the tangential method of x-ray treatment has almost eliminated this trouble. Irradiation of the apex of the lung is inevitable in every case and one occasionally gets a mild degree of the trouble from this, when there has previously been disease at this point.

At first I used strips of lead and limited the beam from an open field by this means, but later I developed a special applicator.

Applicator.—This (Figs. 4, 5, and 6) in its final form has an opening 30 × 12 cm., but the beam is decentered so that the central ray is 1 cm. from the straight side and 11 cm. from the oblique side. The central ray is not actually along the straight side, in order not to cut off rays from a broad focal spot in the tube. It will be realized that since the applicator is lined with lead or lead-rubber, the rays will not enter the body quite at the point where the edge of the straight side touches the skin, but about 1 or 1.5 cm. away. This can be avoided by shaping the applicators like those of Holfelder. Also, the greatest care must be taken to see that the rays go deeply enough into the superficial parts of the lung

Fig. 3. Patient with mediastinal Hodgkin's disease who was treated by various fields directed on to the mediastinum and remained well until he got pneumonia in the right side about a year after the treatment. Skiagram taken four years after the beginning of treatment shows marked bronchiectasis, much displacement of the heart, aorta, trachea, and diaphragm. Since this skiagram there has been considerable improvement in symptoms, but only slight improvement in the radiographic appearance.

to insure sufficient back-scatter to irradiate the deepest parts of the growth. The breast, axilla, and subclavicular regions are included in the fields.

Technic.—For the antero-internal field, will be about 20 to 25 cm. and a tilt of from 10 to 15 cm. in the fields toward the tangent is usually necessary.

The supraclavicular region must be treated in every case and it is not necessary

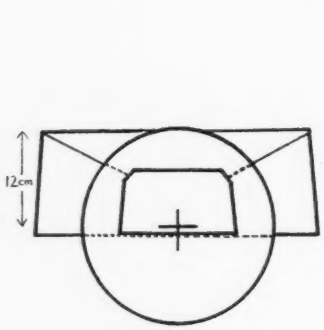

Fig. 4.

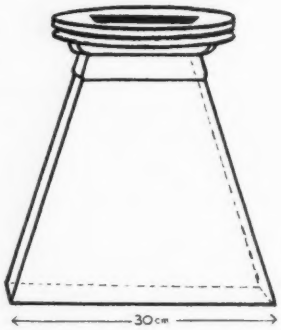

Fig. 5.

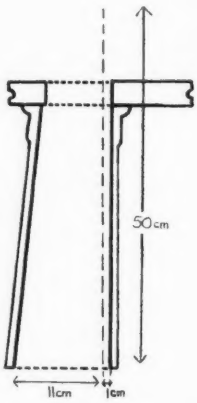

Fig. 6.

Figs. 4, 5, and 6. The writer's special applicator.

the patient lies on the back with arms to the side, and a line is drawn to mark the incident line of the x-rays: the straight side of the applicator touches the skin 1.5 cm. from this. The space which lies between the upper part of the applicator and the tissues is built up with bags of powder of the same density as water: this usually consists of kaolin and sodium bicarbonate mixed in suitable proportions. The line of incidence is made semipermanent by my skin-ink ferric pyrogallate solution in alcohol and acetone, the formula for which is as follows:

Ac. Sulphuros, m. 5
Ac. Pyrogall, gram 1
Acetone, c.c.10
Liq. Ferri Chloridi U.S.P., c.c. 4
Sp. Meth. Industrialis ad., c.c. 20.

For the postero-external field, the patient lies on the side opposite to the lesion with both arms in the front. Sandbags are so arranged as to form a bridge over the lower arm and support the arm of the affected side in a horizontal position. The distance between the centers of the treatment edges

to go through the steps in the evolution of the present method of doing this. What I do, is to give a field directed toward the mediastinum downward and inward to the neck, taking in a region well up the neck.

If palpable hard glands are present above the clavicle, quite a different method must be used. In such a case, the upper mediastinum is undoubtedly invaded by the disease, even if no enlarged glands are seen in the anterolateral skiagram: the outposts of the disease are always ahead of anything which is palpable. In these cases, the upper mediastinum must be treated by multiple ports of entry notwithstanding the possibility of lung changes afterward. The tangential treatment of the pectoral region would be wrong in such a case, as the pectoral skin will be required for the ports of entry into the mediastinum.

I had one case in which both supraclavicular regions contained hard metastatic glands and the mediastinum also showed enlarged glands in the skiagram. This case has remained well now for 14 years after the treatment without any lung changes and I have several other such cases

which have had prolonged periods, from five to ten years, of freedom from disease.

To return to the routine treatment, the incident dose D, at 350 kv. interrupted current and a Thoraeus filter is from 250 to 350 r per day for five days a week and extends from four to five and one-half weeks. The total incident dose, D, is from 2,100 to 3,000 r. The skin dose, D_o, is from 2,600 to 3,750 r. The tumor dose, D_x, is difficult to calculate but is approximately from 3,500 to 4,500 r, except in the subclavicular region and apex of the axilla, where a greater dose is needed and is, therefore, given. The dosage rate is from 18 to 20 r/min. Probably our meter reads low and rather more than this is given.

With 200 kv. continuous current and a Thoraeus filter, we reckon, at St. Bartholomew's Hospital, to give 2,250 r in 18 days, 3,200 r in 24 days, and about 3,800 r in four weeks at the point of lesion.

The inter-field distance varies from 16 to 26 cm. and the surface dose is calculated accordingly.

At the end of the treatment there is a sharp erythema just short of blistering, if the skin is kept dry. This increases for a few days and then subsides and only in rare instances causes subsequent trouble.

Differences from Holfelder's Method.—In this method the applicator is much wider and consequently the lungs tend to receive more radiation, as the rays from the patient's side of the applicator are much more oblique (Figs. 7 and 8).

I originally treated the external area with the arm drawn back but I now draw the arm forward and place the applicator behind it, as Holfelder does. With the much wider field (25 × 20 cm.) which he uses, Holfelder gets more back-scatter from his bolus, while I rely more on direct radiation. Apart from these differences, the methods have the same object and are essentially the same.

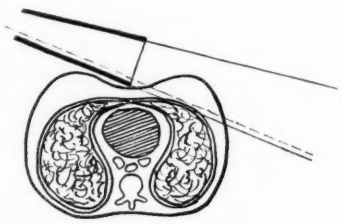

Fig. 7.

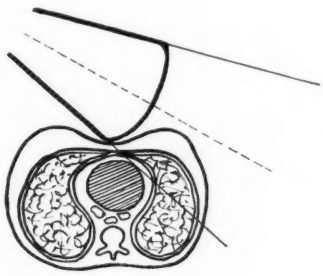

Fig. 8.

Figs. 7 and 8. Differences between the Finzi and the Holfelder applicators (slightly exaggerated).

Other Applications.—For certain other regions, such as a one-sided skull-bone tumor, especially if the eye can be avoided by an oblique tangent, for a localized parotid tumor, for growths of the pinna, and so on, other applications have proved useful.

In conclusion, I may say that the tangential method is the one which we use as routine in suitable cases and it has given us complete satisfaction, the only disadvantage being that it requires anatomical knowledge and extreme care in application. The only contra-indications are involvement of the supraclavicular or mediastinal glands or the occurrence of distant metastases. Previous irradiation compels very serious consideration as to dosage and whether any further irradiation is justifiable.

CHRONIC RECURRENT INTESTINAL INTUSSUSCEPTION[1]

By MILTON G. WASCH, M.D., Attending Radiologist, Jewish Hospital and Israel Zion Hospital, *and* BERNARD S. EPSTEIN, M.D., Assistant Radiologist, Jewish Hospital, *Brooklyn, New York*

ALTHOUGH chronic recurrent intussusception of the large bowel is uncommon, it must be considered in the differential diagnosis of obscure intra-abdominal conditions. The writers have had the opportunity of observing eight cases diagnosed radiographically and confirmed by operation. Each patient was examined by means of a barium meal from above, as well as by opaque enemas, post-enema radiograms, and barium-air contrast enema radiograms. Repeated examinations were made in several instances. The post-evacuation and barium-air contrast enema studies were so helpful in establishing the diagnosis that we now regard them as indispensable.

Seven patients had cecocolic intussusception secondary to cecal tumors. In these cases, the ileum also intussuscepted. Pathologic examination revealed five adenocarcinomas and two leiomyosarcomas. The eighth patient had a colocolic invagination associated with a pedunculated leiomyosarcoma arising from the hepatic flexure.

The association of chronic large bowel intussusception with leiomyosarcoma is unusual. We have been unable to find reports of similar cases in the available literature.

Symptomatology.—The clinical picture of the seven patients with ileocecocolic intussusception was essentially the same. All complained of more or less persistent pain in the right lower quadrant, radiating to the epigastric and substernal regions. The pain varied considerably in intensity at different times. Nausea and anorexia were frequent. Vomiting and bloody stools were often observed. There were relatively long intervals of freedom between the acute episodes, and cathartics on occasion aggravated the symptoms. The history presented by the patient with colocolic invagination was somewhat more acute, only one attack of persistent right upper quadrant pain occurring before surgical intervention.

The chronicity of the symptoms, repeated subacute episodes, and the changing radiographic findings are in contrast with the syndrome usually observed in the acute forms of intussusception.

The relationship between the size of the intussuscipiens and intussusceptum no doubt plays an important rôle in the severity of the symptoms. In this connection Ehnmark (1) recalls that the large and small bowel of children are approximately the same size, whereas the large bowel of an adult is about three times as large as the small. Hence an intussusception in an adult involving the presence of ileum within the large bowel may be expected to produce symptoms of less severity than a colocolic invagination. This is supported by the findings in the cases here reported, in which the patients with ileocecocolic invagination had relatively chronic courses while the patient with colocolic invagination had but one attack before operation. In the latter case, it is possible that the presence of a long pedicle attached to the tumor mass helped produce more acute symptoms inasmuch as the constriction of the bowel about the pedicle prevented recession of the intussusception.

In the patients with ileocecocolic intussusception, the principal finding was a soft doughy tender mass in the right abdomen unaccompanied by rigidity.

In the case with colocolic invagination, starting at the hepatic flexure, the mass was palpated in the epigastrium. In most in-

[1] This is one of a series of papers contributed by friends and former pupils of I. Seth Hirsch, M.D.

stances, the tumor possesses spontaneous and passive mobility except when fixation occurs as a result of peritoneal reaction. In these cases, abdominal rigidity is usually present. The mass disappears with release of the intussusception, only to reappear with a recurrence.

Radiology.—The radiographic diagnosis of recurrent intussusception was first made by Snow and Clinton (2), in 1913. Frequent reports since have stressed the radiographic appearance (3, 4, 5, 6), as well as the therapeutic possibilities offered by barium clysmas (7, 8). Recently, Medo (9) published illustrations of various configurations resulting from invagination of the large bowel.

A consideration of the anatomy of intussusception is essential to the understanding of the radiographic picture. There is no single pathognomonic sign, the roentgenographic appearance varying with the degree and location of the intussusception at the time the examination is made. If the intussusception is sufficiently large, it may block the progress of the barium completely. Lesser degrees of obstruction may cause the barium to be trapped in the rugal folds, resulting in a wide range of roentgenographic patterns.

The anatomic changes produced by the intussusception may result in ballooning of the bowel proximal to the obstruction, into which the barium clysma may be unable to enter; or the barium may trickle through, string-like, into the distended arm. In other instances, the advancing head of the intussusceptum may partially obstruct the barium stream, producing configurations which have been referred to as "pincer-like," "beak-like," "cupola-like," "spiral," or other descriptive terms. (The composite finding of several examinations is more suggestive than any single observation.) No annular constriction nor worm-eaten irregularity such as is often observed with infiltrating neoplasms of the large bowel is present.

The barium-air contrast studies often portray a tumor by curvilinear barium markings in relief against the air-distended bowel. The tumor mass is thus often seen in silhouette sparsely covered and streaked

Fig. 1. Case 1. Post-enema air inflation study showing cecal tumor with characteristic curvilinear striations independent of normal haustral markings.

with barium as it rests within the crevices of the neoplasm.

Case Reports.—Case 1. L. F., a 58-year-old white housewife, complained of vague abdominal pains above the right ilium for several years. The pains had increased in severity during the three weeks previous to admission, were intermittent non-radiating in character, and varied considerably in intensity. Gradual loss of weight had been noted. About ten days before admission, the patient had had an acute attack of right lower quadrant pain "after eating prunes," which lasted several hours. There was no nausea or vomiting.

Physical examination revealed an oval tender mass in the right abdomen at the level of the iliac crest. There was no abdominal rigidity; visible peristalsis was present. On successive occasions the mass appeared and disappeared. Examination of the stool revealed the presence of occult blood.

Radiographic examination of the large bowel by means of a barium enema was not conclusive. Air inflation, after the

evacuation of the enema, clearly revealed the presence of a cecal tumefaction. The radiographic appearance of the ascending colon varied at successive examinations.

Fig. 2-*A* (*upper left*). Case 2. Barium enema demonstrating concentric striated defect of cecum fusing with the hepatic flexure due to partial intussusception.

Fig. 2-*B* (*upper right*). Case 2. Contrast enema study demonstrating reduction of incomplete intussusception by air pressure, and clear visualization of the tumor in the cecum with classical defects.

Fig. 3-*A* (*lower left*). Case 3. Post-enema study demonstrating intussuscepted tumor mass in the hepatic flexure.

Fig. 3-*B* (*lower right*). Case 3. Barium enema eight days later demonstrating tumor in the cecum with the intussusception spontaneously reduced. At operation the intussusception was found to have recurred.

At operation, a mass extending from the middle of the ascending colon to the hepatic flexure was found. The tumor was bluish in color and contained an invaginated section of ileum which could not be reduced. The patient died the following day.

An autopsy, a fungating cauliflower-like mass was found in the cecum, extending to and involving the ileocecal valve. Histologic examination of the mass revealed adenocarcinoma.

Case 2. B. J., 30-year-old male, complained of persistent right lower quadrant pain, marked anorexia, and vomiting for seven weeks. Pains occurred from two to eight times daily and lasted but a few seconds. The symptoms had appeared gradually and varied considerably in intensity. Some relief had been obtained from bicarbonate of soda. There was no febrile reaction. The patient had lost 35 pounds in weight in six weeks before hospitalization. Blood had been noted in the stool.

Physical examination revealed a moderately tender mass deep in the right lower quadrant, which did not change in size during the pre-operative period of observation. There was no abdominal rigidity.

A barium enema revealed shortening of the ascending colon and an obliteration of the angle of the hepatic flexure. A filling-defect was present in the cecal region. An air-contrast radiograph definitely showed cecal tumefaction.

At operation, an intussusception of the cecum and ascending colon into the transverse colon was demonstrated. A cauliflower-like growth about six centimeters in diameter, arising from a short pedicle, was present in the cecum. The mass was soft to the touch and worm-like in appearance. The intussusception was reduced and the tumor excised.

Histologic examination of the specimen revealed a leiomyosarcoma. The pathologist's description is as follows: "The mucosal lining of the papillary structures show very marked hyperplasia of the mucous glands. No evidence of invasive changes of any epithelial elements is noted. The connective tissue and muscular coats show marked proliferative changes, and at the base of the pedicle of each of the papillary masses may be found large spindle-shaped cells, the nuclei of which show marked activity and in general fulfill the criteria for malignant change. Most of the spindle-shaped cells can be definitely seen arising from the muscular layer."

Case 3. L. F., a 59-year-old housewife, complained of intermittent colicky pain in the right lower quadrant, radiating to the epigastrium and substernal regions. Episodes of this character had occurred infrequently for about two years. They had increased in frequency during the six weeks previous to admission to the hospital. The onset of pain was gradual, and on occasion followed a cathartic. Attacks were often preceded by diarrhea and bloody stools. Anorexia was present, and there had been considerable loss of weight.

Physical examination revealed a soft doughy mass in the right lower quadrant which appeared and disappeared on different occasions. No abdominal tenderness, rigidity, or visible peristalsis was present.

A barium enema revealed a dome-shaped obstruction at the hepatic flexure with a large gas defect distal thereto, through which some barium trickled, visualizing false cecal rugal markings. An immediate post-enema study clearly portrayed a mass in the proximal portion of the transverse colon. A barium enema one week later readily revealed cecal tumefaction, and the barium-air contrast radiograph more clearly outlined the tumor.

At operation about one month later, an ileocecocolic intussusception was found due to a sessile mass in the cecum which involved the lower lip of the ileocecal valve. Pathological report was that of adenocarcinoma.

Case 4. S. R., a 48-year-old male, complained of persistent dull-aching right lower quadrant pain, varying in intensity for six weeks. Tarry stools had been present on several occasions. There was no loss of weight, anorexia, vomiting, nor fever.

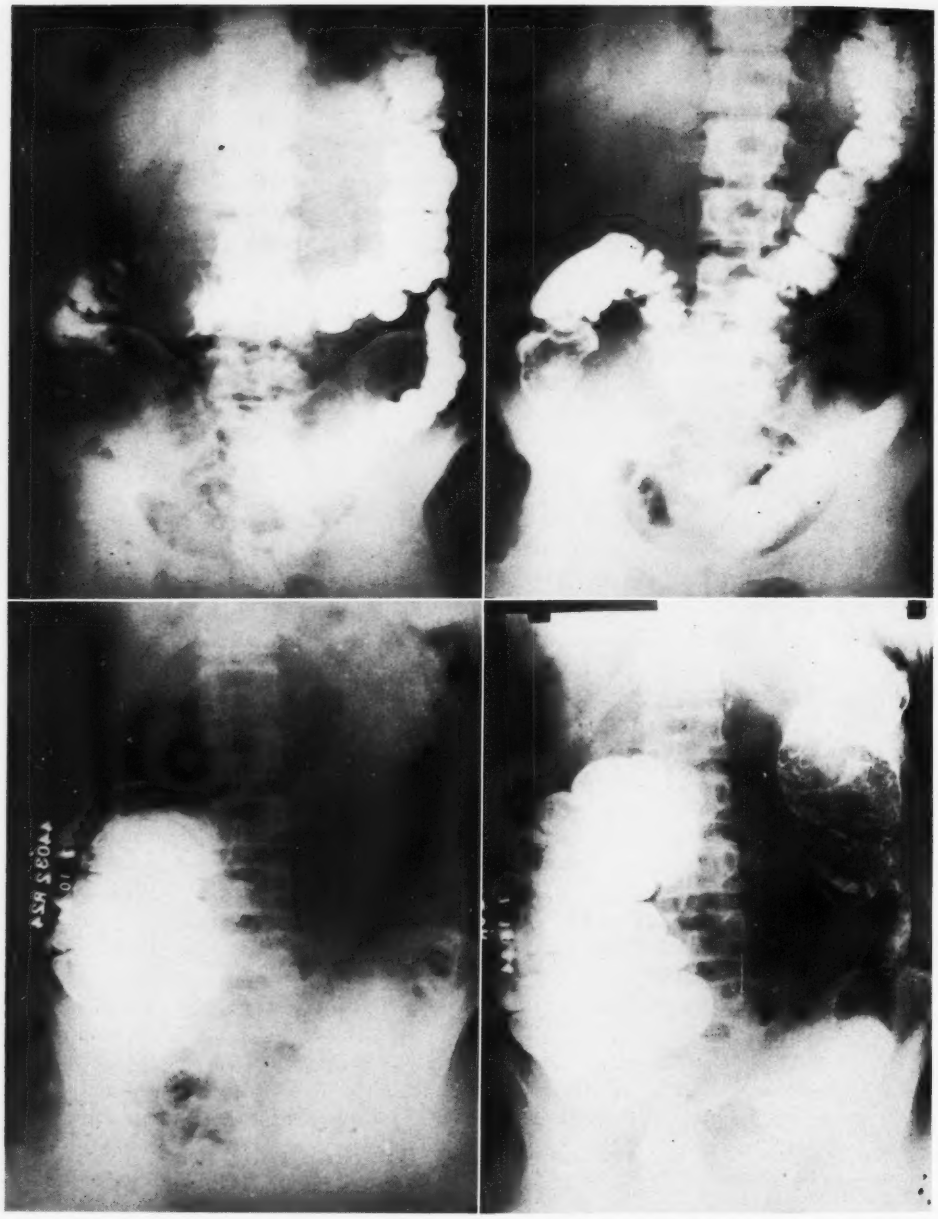

Fig. 4-*A* (*upper left*). Case 4. Twenty-four-hour barium meal study demonstrating defect just beyond the hepatic flexure, associated with localized gaseous distention.

Fig. 4-*B* (*upper right*). Case 4. Post-enema study showing deformity now in cecal region. The filling-defect at the hepatic flexure is no longer present.

Fig. 5-*A* (*lower left*). Case 5. Twenty-four-hour study showing barium meal trapped in ascending colon, with marked distention.

Fig. 5-*B* (*lower right*). Case 5. Barium-air contrast enema six days later still showing barium trapped in ascending colon. Hiatus in transverse arm due to intussuscepted bowel with intraluminal neoplasm beyond. Note characteristic concentric striations.

Physical examination revealed tenderness in the right lower quadrant. Some resistance to deep palpation was noted but pain eased within a few hours, then returned and persisted for two weeks. No similar episodes had occurred previously.

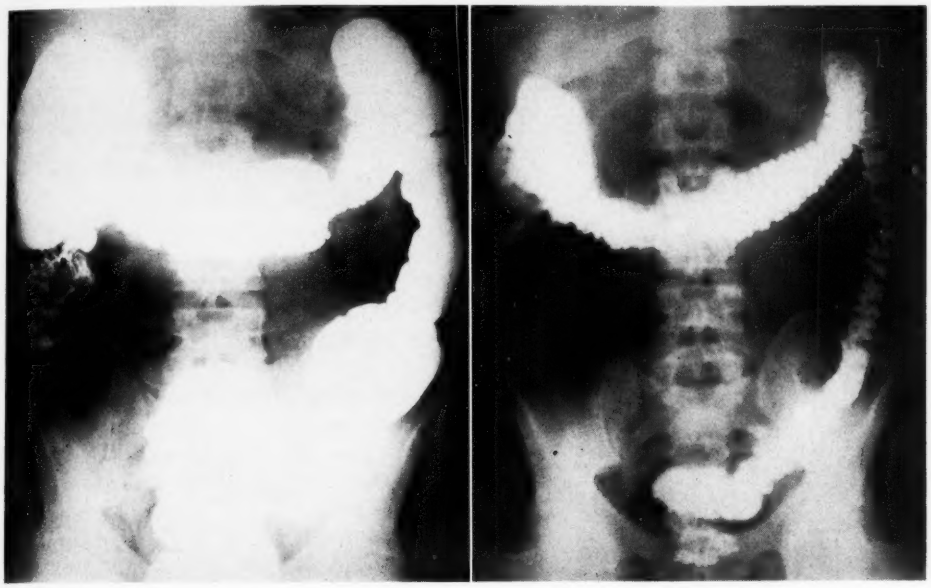

Fig. 6-*A*. Fig. 6-*B*.

Fig. 6-*A*. Case 6. Barium enema showing filling-defect in cecum with honey-comb markings as the barium dips into the tumor crevices.
Fig. 6-*B*. Case 6. Post-enema filling showed intussuscepted tumor mass at the hepatic flexure.

no true abdominal rigidity. A questionable mass at times was palpated.

X-ray examination 24 hours after a barium meal revealed elevation of the cecum, with a filling-defect in the hepatic flexure distally. A barium enema flowed freely from rectum to cecum. After evacuation of the enema, however, a cupola-like deformity was present proximal to the hepatic flexure, with distortion of the normal cecal contour.

At operation, a sessile mass about eight centimeters in diameter was found in the cecum. The ileocecal valve was involved. It apparently had been intussuscepted and reduced spontaneously previous to operation. Histologic examination of the specimen revealed adenocarcinoma.

Case 5. M. A., a 55-year-old housewife, complained of pain in the right upper quadrant after taking a cathartic. The pain eased within a few hours, then returned and persisted for two weeks. No similar episodes had occurred previously. A tarry stool was noted for the first time a month before admission to the hospital. There was evident loss of weight. No anorexia, vomiting, nor diarrhea had been noted.

Physical examination revealed a very tender large nodular mass in the epigastric and umbilical regions. The tumor was constant and did not vary in size.

A film taken 24 hours after a barium meal revealed the barium trapped in the ascending colon, which was markedly distended. The barium remained there for six days. An enema then administered flowed to the hepatic flexure, but not beyond. A post-enema air-barium contrast radiograph brought the mucosal markings of the transverse and descending arms into relief. Some barium appeared to have reached the ascending colon. The marked irregularity adjacent to the obstruction was

interpreted as evidence of intrinsic pathology. A hiatus in the transverse colon was present on all films.

Fig. 7. Case 7. Intussusception arising from perforating carcinoma at hepatic flexure. Adjacent duodenum was involved and peritoneal implants were present. A contrast enema would probably have visualized the tumor more clearly.

At operation, an intussusception of the hepatic flexure into the transverse colon was found. A polyp about five centimeters in diameter arising from a long slender stalk in the hepatic flexure was the head of the intussusception. The invagination was reduced and the polyp removed. Histologic examination of the polyp revealed a leiomyosarcoma.

Case 6. D. R., a 42-year-old male, was admitted to the hospital complaining of having had intermittent abdominal cramps, diarrhea, and bloody stools for nine months. The onset had been gradual, usually occurring one or two hours after meals. At first cramps were generalized and later were located in the right lower quadrant. There were no chills nor temperature. Bright red blood was first noted in the stool three months before admission; tarry stools likewise were noted. There was no constipation. The patient had lost 16 pounds in weight since the onset of symptoms.

On physical examination some tenderness was noted in the lower quadrants along the ascending arm and sigmoid. No mass could be palpated.

X-ray examination by means of a barium enema revealed a filling-defect in the cecal region. The caput rested at the iliac crest. The rugal markings of the cecum were bizarre and stood out in relief against the filled bowel, separated from it by a definite line of demarcation. After evacuation of the enema, the cecum still remained elevated.

At operation, a hard indurated mass was found in the cecum, and about four inches of intussuscepted ileum was present. Histologic examination of a biopsy specimen revealed a papillary colloid adenocarcinoma.

Case 7. L. W., a 50-year-old male, complained of abdominal pains and progressive weakness for the past four months. Rectal bleeding at times was attributed to hemorrhoids (?), although a hemorrhoidectomy had been performed two years previously. Diarrhea was present for four days prior to admission. The patient vomited on two occasions. His temperature was from 101 to 102°. There was belching, nausea, and peri-umbilical distress. Physical examination of abdomen was negative. Blood count revealed: red blood cells, 3,200,000; hemoglobin, 40 per cent; white blood cells, 5,900. The urine was negative.

A barium enema disclosed an obstruction at the hepatic flexure, with telescoping of the cecum and ascending arm into the transverse colon.

Operation revealed a mass at the hepatic flexure, fixed posteriorly, produced by an intussusception of the cecum and ascending colon into the hepatic flexure and adjacent portion of the transverse arm. The mass was adherent to the duodenum, which presented carcinomatous implants on its serosa. Surrounding exudate was pres-

ent. Following reduction, a bulky tumor mass was found in the cecum.

Pathologic diagnosis was: ulcerated adenocarcinoma of cecum, with perforation and localized abscess formation.

Though the x-ray in this case was diagnostic, the tumor mass itself was not demonstrated. It is our belief that had a contrast enema been given, in addition to the intussusception, the tumor mass would have been visualized.

Case 8. D. K., a female 57 years of age, suffered from recurrent febrile reactions, ranging from 100 to 103° for the past several weeks. There was general weakness and constipation so that daily enemas were employed. Three days previous to admission, there was transient epigastric distress but no vomiting. Her appetite was fair at times only. Mucus and blood were found in a diarrheal stool nine days after admission. From this time on, blood was found on repeated occasions. There was no palpable mass, distention, nor abdominal rigidity. Slight tenderness on the right side of the abdomen was present at times.

A barium clysma flowed freely through the bowel to the hepatic flexure where it met with an obstruction. The ascending arm was not successfully outlined and no tumor mass was demonstrated. Intussusception was suggested from the barium contrast enema skiagram, which revealed a large irregular tumor mass at the hepatic flexure. The deformity from the tumor itself was characteristic—a negative, somewhat globular defect, with curvilinear barium striations throughout, in a grossly distended bowel.

Operation revealed a mass at the hepatic flexure consisting of intussuscepted terminal ileum and cecum. After reduction, which was relatively easy, a palpable sessile mass, about three centimeters in size, was found in the cecum. The pathologic report was a circumscribed papillomatous tumor which, on section, proved to be a leiomyosarcoma.

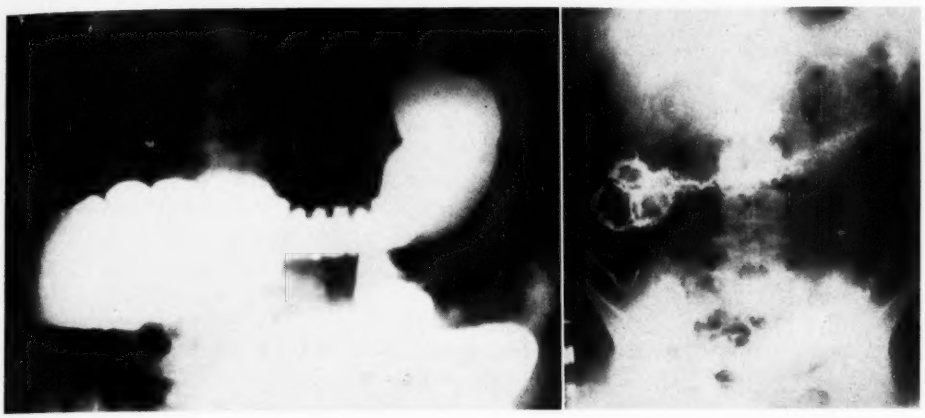

Fig. 8-*A*. Fig. 8-*B*.

Fig. 8-*A*. Case 8. Barium enema. Ileocecal intussusception from cecal tumor. Note dilatation of bowel embracing the intussusceptum. Cecum telescoped and not visible.

Fig. 8-*B*. Case 8. Contrast enema study demonstrating large intussuscepted tumor at the hepatic flexure. The intussusception has reduced partially following the procedure.

CONCLUSIONS

1. Eight cases of chronic intussusception of the large bowel in adults are reported.

2. Subacute episodes, with varying intervals of quiescence, are the rule.

3. Repeated x-ray examinations of the colon are important in diagnosis.

4. Post-evacuation radiographs and barium-air contrast enemas have proved the most valuable methods of study in our hands.

REFERENCES

(1) EHNMARK, E.: Intestinal Intussusception at Cecal Tumors. Acta chir. Scandinav., **76,** 147–211, 1935.

(2) SNOW, I. M., and CLINTON, M.: The Diagnosis of Intussusception by the X-ray. Am. Jour. Dis. Child., **6,** 93, 1913.

(3) SUSSMAN, MARCY L.: Roentgenologic Aspect of Subacute and Chronic Intestinal Intussusception. Am. Jour. Roentgenol. and Rad. Ther., **27,** 374–382, March, 1932.

(4) LAURELL, H.: Ueber reine Coloninvaginationen vom roentgenologischen Gesichtspunk. Acta Radiol., **14,** 122, 1926.

(5) GEULETTE, R.: De l'invaginatione intestinale, ses formes cliniques chez l'adult. Presse méd., **34,** 1226, 1926.

(6) GASTRONOVO, E.: Gli Aspetti Radiologic della invaginazione ileocecale subacuta e cronica. Radiol. Med., **16,** 917, 1929.

(7) STEPHENS, V. R.: Acute Intussusception, Manipulative Reduction under Fluoroscopic Control. Am. Jour. Dis. Child., **35,** 61–64, 1928.

(8) SJÖSTROM, P. M.: Ueber Diagnostik und Disinvagination von Darminvaginationsfällen mit Hilfe von Röntgendurchleuchtung. Acta chir. Scandinav., **74,** 125–171, 1934.

(9) MEDO, G.: Studio Radiologica dell' invaginzione intestinale. Radiol. Med., **16,** 821, 1929.

THE ANATOMICAL POSITION OF THE ILEUM IN HEALTH AND DISEASE[1]

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IT IS NOW almost six years since the attention of the medical world was attracted to the subject of regional or, more often, terminal ileitis. In that short space of time, the full clinical picture has been identified, the etiology discussed, the complications noted, and the surgical therapy amply instituted.

The fact that any part or segment of the ileum might be, and often is, involved, and the recognition of participation of higher sites in the jejunum have led to the name "regional enteritis." Yet for practical purposes the main brunt of the disease, and by far its most common form, is not a general enteritis, but a terminal ileitis, the last six to twelve inches of ileum being exclusively involved in over 90 per cent of all cases.

In addition, a common complication of fistula formation frequently characterizes the disease, these fistulous tracts making their terminus in the lower right anterior abdominal wall or traversing the pelvis to appear in the perineum as perirectal or recto-vaginal fistulas.

If one were to ask an anatomist, an internist, a surgeon, or even a radiologist the normal position and site of the terminal segment of the ileum, the answer would likely be hesitant, if a correct one, and would probably be based more on a general impression than upon accurate knowledge or observation.

The sparsity of accurate data regarding the true anatomical position of the terminal ileum and the study of these low fistulous complications have together led us to attempt to ascertain the exact position of the terminal loop of ileum and its anatomical relationship, both in the normal body and when it is the object of a disease process.

Anatomical and Embryological.—References to the exact position of the terminal ileum are sparse in anatomical textbooks and practically missing from current literature. Very few anatomists mention the subject at all; those who do—Testut, Sernoff, Piersol—place the terminal ileum deep in the pelvic fossa on the right side, though exact observations or notations are brief and insufficient from a practical standpoint.

The exact anatomical location of the terminal ileum is dependent upon the normal embryological development of the ileocecum and its associated mesenteries. Ontogenetically, the ileocecum develops from that portion of the midgut which is caudad to the superior mesenteric artery, "the post-arterial segment" of Dott; according to Mall and to Huntington, the ileocecum is returned to the celomic body cavity from the physiological umbilical pouch during the tenth week of embryonal development. It assumes primarily an anterior position beneath the right lobe of the liver whence it descends to the normal adult position in the right iliac fossa. The mesentery of the ileum is formed by the fusion of the superior mesenteric artery to the posterior abdominal wall; this mesentery becomes, and is, continuous with the mesentery of the ascending colon. Both of these processes are usually effected before parturition but they may be delayed until after birth.

According to Alglave, the cecum fails to descend, remaining high under the liver in almost 3 per cent of all cases. On the other hand, it may descend to the other extreme, that is, below the true pelvic brim in about 10 per cent of 1,050 specimens studied by G. M. Smith. In this latter series, the cecum and presumably the terminal ileum were found resting on the

[1] This is one of a series of papers contributed by friends and former pupils of I. Seth Hirsch, M.D.

pelvic diaphragm in contact with the bladder and the rectum in 2 per cent of the cases examined.

Testut, in his anatomical studies, places the cecum below the pelvic inlet in 15 per cent of male and in 30 per cent of female bodies.

To a large degree, the position of the terminal ileum depends upon the position of the cecum, which latter again depends, to a certain extent, upon the existence or non-existence of the mesentery of the ascending colon. If the ascending colon is fully peritonealized with an ample mesentery, which occurs in 26 per cent of cases (Treves), the cecum is likely to hang low in the iliac fossa or over the brim of the pelvis. If it possesses no mesentery, it will likely be found fixed and firm in the iliac fossa above the brim of the true pelvis.

In 90 per cent of cases, the cecum is fully surrounded by peritoneum; in 10 per cent it is more or less adherent in the false pelvis (Morris).

The attachment of the mesentery of the small intestine begins above and to the left of the first lumbar vertebra, descending obliquely to the right sacro-iliac level whence it passes transversely and fuses with the mesentery of the colon. The terminal loops of ileum hence are attached at their mesentery to the brim of the true pelvis and fall from before gracefully in vertically placed loops backward in the pelvic chamber. The position of these terminal segments of ileum is relatively fixed. The segments reassume their position after laparotomy (Cruveilhier, Gregoire).

In the upright or standing position, these loops fall into the space between the bladder and the rectum (pouch of Douglas) in the male, or between the posterior surface of the uterus and the anterior surface of the rectum in the female. Very often these loops lie directly on the lowermost point of the pelvic floor, in the cul-de-sac actually between the posterior wall of the vagina and the rectal surface behind.

Monks found the terminal ileum to be in the pelvis in more than 50 per cent of his studies; Mall states that normally the terminal loop of ileum rises from the pelvis into the right iliac fossa to join the mesial aspect of the cecum.

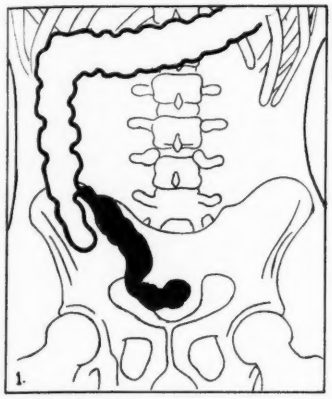
The position of these loops is fairly constant; fecal impaction of the rectum and sigmoid and an overfull bladder may push the terminal ileum out of the pelvis; enlargements of liver and spleen, intra-abdominal masses, and generalized visceroptosis will push it further into the pelvis.

In most textbooks and publications on roentgenology, it is difficult to find exact statements as to the position of the terminal ileum. Schinz, Baensch, and Friedl state that the last loop of the ileum normally rises out of the pelvis and opens into the mesial side of the cecum. Occasionally the last ileal loop is adherent to the side of the pelvic wall.

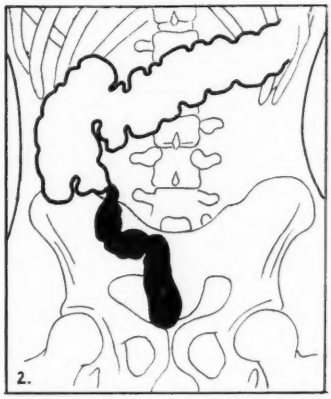
To determine by radiography the usual position of the normal ileum and its variations, we studied 150 control cases, both by barium meal and barium enema, accepting only such cases in which the terminal ileum was clearly demarcated as to its position and course. Cases were chosen in which no organic disease of the intestine was suspected. All plates were taken in the prone position, 3- to 6-hour plates being chosen for the meal method, and evacuation plates in the enemas. Occasionally plates were also taken in the standing position to determine differences in the site and the course of the ileum in the contrasting positions. The group consisted of 77 females and 73 males.

The position of the insertion of the terminal ileum into the cecal wall was chosen as a constant, and is illustrated in Table I. The figures in this table agree in a general way with the observations of Kantor, who utilized the top of the acetabulum as a fixed point from which to measure the position of the cecum.

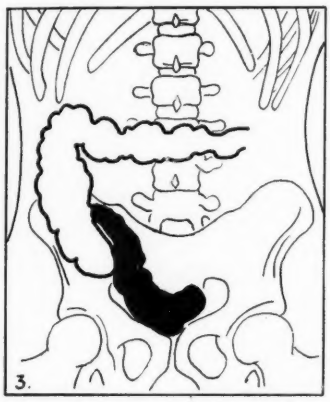
In 18 per cent of the cases, the cecum was actually found to be in the pelvis; in one case, practically the entire small intestine plus the ascending colon and mid-transverse colon appeared to be within the true



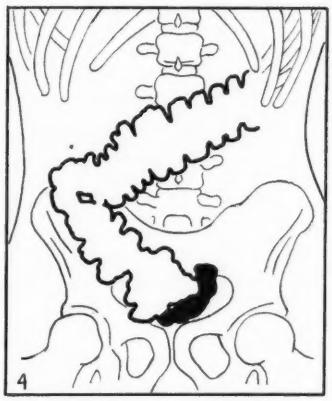
Usual position of cecum + ileum



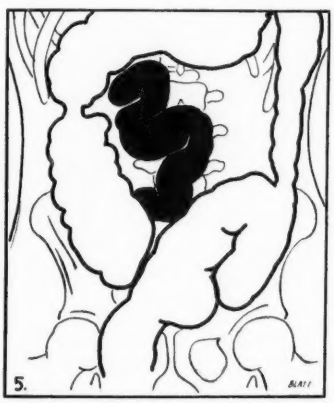
High cecum-ileum to pelvis



Low cecum-high medial implantation of ileum



Complete descent-cecum + ileum in pelvis



Exceptional position of terminal ileum-abrupt ascension

NORMAL TERMINAL ILEUM – ANATOMICAL POSITIONS

Plate 1.

pelvic cavity. The splenic flexure was situated at the level of the iliac crest.

The course and site of the terminal ileum was well visualized in these 150 cases. In 72 per cent, the terminal segment of the ileum arose from the cavity of the true pelvis, where it lay on the floor of the pelvis, to ascend and join the medial aspect of the cecum in the right iliac fossa (Fig. 1, Plate 1). When the cecum rides high, the course of the terminal ileum is the same but the ascent is greater (Fig. 2, Plate 1). In the instances of low implantation of the cecum, where its tip dips into the true pelvis, the course of the ileum is more circuitous, the ileum curving around the medial aspect of the cecum to a rather high implantation at the ileocecal junction (Fig. 3, Plate 1).

TABLE I.—JUNCTION OF TERMINAL ILEUM AND CECUM

Vertebral Body	Case Incidence
4th lumbar	3
5th lumbar	9
5th lumbar to 1st sacral	17
1st sacral	58
1st and 2nd sacral	32
2nd sacral	2
3rd sacral	1
In pelvis	28
Total	150

In 26 per cent of the cases, the cecum was well within the true pelvic cavity. In these instances, the entire terminal loop of the ileum as well as the ileocecal junction appeared to lie directly on the pelvic floor (constituted by the levator ani muscles, Fig. 4, Plate 1). In only three cases (2 per cent), the terminal segment of the ileum was not at all in the pelvis but arose high from its junction with the cecum, passed transversely for from one to two inches, and was then directed upward toward the transverse colon. This unusual direction was demonstrated by both meal and enema methods. The loop could not be depressed nor its position changed by manual pressure under fluoroscopic control (Fig. 5, Plate 1).

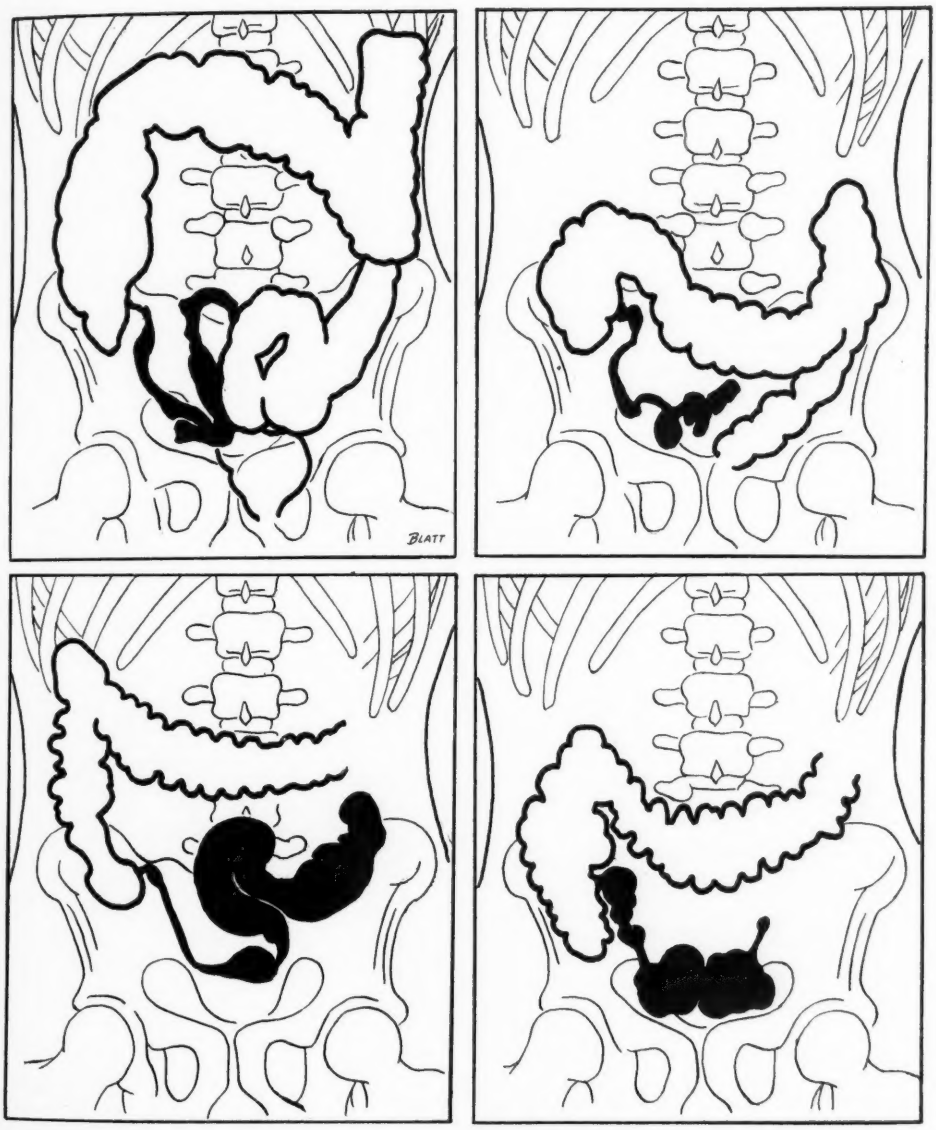
Pathological Ileum.—The plates of 25 cases of "terminal ileitis" were studied for the position of the diseased segment. In all of these instances, the pathological terminal loop lay within the true pelvis; in fact, on the floor of the pelvic cavity, its course as in the normal control cases, upward to its entrance at the ileocecal junction. This course and site is practically uniform. In the diseased instances, the ileocecal junction is regularly at or below the level of the first sacral vertebra (usually fixed by adhesions) and the terminal inches of the ileum descend directly to the pelvic floor.

In one instance, the diseased segment made a short horizontal course and then descended into the pelvis; in another case with more extensive involvement of the whole ileum, the diseased loop was seen to descend to the floor of the true pelvis and then to rise out of the pelvis toward the left mid-abdomen. This latter course is true of all cases of segmental enteritis in which more than the terminal loop is affected; here the more proximal diseased areas occupy the lower, mid-, or right abdomen, ascending as the process extends toward the left, mid-, or upper abdomen.

In several instances, we have true and accurate surgical descriptions of the site and position of the diseased terminal ileum in which the terminal segment was found to be in the true pelvis. The entire pelvis was found to be occupied by a mass of matted small intestine, usually adherent to the peritoneal reflexion over the sacrum, to the base of the bladder, or to the sigmoid by firm dense adhesions. Occasionally even the tip of the omentum was found to be in the pelvis adhering to and covering the diseased segment. In one accurate description by an observing surgeon, it is stated that the cecum was bound down and fixed by an inflammatory exudate arising from the ileum. The terminal ileum descended from its fixed point of attachment to the cecum, extending abruptly downward into the cul-de-sac behind the uterus and adherent to the anterior rectal wall. The diseased ileum was not removed; four months later the patient developed a fecal recto-vaginal fistula.

Remarks.—In 15 per cent of our observed clinical cases of ileitis, we have noted perineal fistulas (perirectal, perineal, or rectovaginal) as a complicating factor. In several of these cases (Penner and Crohn, Crohn), we had reason to believe that a direct fistulous tract made its course from the diseased loop of ileum lying on the floor of the true pelvis, seeping downward through the fascial planes to make its exit somewhere on the perineum between the rectum and the vagina and involving either one or both of these organs.

In order to establish this hypothesis, it



TERMINAL ILEITIS - ANATOMICAL POSITIONS

Plate 2.

was necessary to determine the true position of the terminal ileum, first in health, and then when diseased.

The above study confirms these ideas, insofar as it is shown that both in health and under pathological conditions the terminal ileal segments lie almost uniformly on the pelvic floor in direct contact with the peritoneal reflexion and fascial planes covering the levator ani suspensory muscles.

This position of the normal and diseased terminal ileum as described by those few anatomists who have made actual notations, has been confirmed by routine roentgenography and has been transcribed in detail by observing surgeons. The findings rationalize the clinical observations of fistulous perineal tracts emanating from the low-lying ileum and making their exit by gravity and by following fascial planes of cleavage onto the perineal floor.

REFERENCES

1. Testut, L., and Latarjet, A.: Traité d'Anatomie Humaine, **4,** 400. Gaston Doin, Paris, 1931.

2. Sernoff: Zur Kenntniss d. Dunndarms. Intern. Monatschr. f. Anat., **11,** 437–466, 1894.

3. Piersol, G. A.: Human Anatomy, p. 1652, J. B. Lippincott Co., Philadelphia and London, 1918.

4. Dott, N. M.: British Jour. Surg., **11,** 251–286, October, 1923.

5. Mall, F. P.: Development of Human Coelom. Jour. Morphology, **12,** 395, 1897.

6. Huntington, G. S.: Anatomy of Human Peritoneum and Abdominal Cavity, p. 58. Lea Bros., Philadelphia, 1903.

7. Alglave, P.: Traitment Chirurgical de la Tuberculose Ileocecal, p. 13, Paris Theses, 1903, 1904.

8. Smith, G. M.: Anatomic Record., **5,** 549, 1911.

9. Treves, Sir F.: Anatomy of Intestinal Canal and Peritoneum. Fourth edition, p. 55. Hunterian Lectures. H. K. Lewis, London, 1885.

10. Morris: Human Anatomy. Edition of C. M. Jackson, p. 1196, P. Blakiston's Son & Co., Philadelphia, 1925.

11. Cruveilhier, J.: Anatomy of Human Body. Third edition, p. 365, Harper & Bros., New York, 1875.

12. Gregoire: Traité d'Anatomie Humaine. P. Poirier and A. Charpy. **4,** Author, T. Jonnesco, second edition, p. 308, Masson & Cie, Paris, 1901.

13. Monks, G. H.: Studies of Surgical Anatomy of the Small Intestine and its Localization. Ann. Surg., **42,** 543–569, October, 1905.

14. von Schinz, H. R., Baensch, W., and Friedl, E.: Lehrbuch der Röntgendiagnostik, **2,** 1341, G. Thieme, Leipzig, 1932.

15. Penner, A., and Crohn, B. B.: Peri-anal Fistulæ as Complication of Regional Ileitis. Ann. Surg., **108,** 867–873, November, 1938.

16. Crohn, B. B.: Surg., Gynec. and Obst., **68,** 314–321, February, 1939.

X-RAY THERAPY IN THE TREATMENT OF ACUTE PNEUMONIA[1]

REPORT COVERING THE USE OF X-RAY THERAPY IN THE TREATMENT OF PNEUMONIA AT THE NIAGARA FALLS MEMORIAL HOSPITAL, FROM OCT. 1, 1937, TO SEPT. 30, 1938

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THE presentation by Eugene V. Powell, M.D., of Temple, Texas, of his paper entitled "Roentgen Rays in the Treatment of Acute Pneumonia" at the Fifth International Congress of Radiology, in Chicago, September, 1937, brought to our attention a use for x-ray therapy which, up to that time, had been almost entirely neglected. Subsequent publications by this author should be reviewed by those interested in the pneumonia problem (1,2).

This neglect of x-ray therapy in such a definitely acute infectious condition as pneumonia seems strange, in retrospect, to radiologists and to other physicians who may be familiar with the value of x-ray therapy in acute infections when we think of the advances in the use of x-ray therapy in the past 35 years.

The use of x-ray therapy in the treatment of boils, carbuncles, erysipelas, cellulitis, gas infection, otitis, mastoiditis, breast abscess, adenitis, parotitis, phlegmon, onychia and paronychia, gonorrheal arthritis, and other infectious conditions may be said to have gone far beyond the experimental stage.

The report here offered is chiefly a summary of 138 cases of pneumonia which passed through the X-ray Department of the Niagara Falls Memorial Hospital during one year, Oct. 1, 1937, to Sept. 30, 1938, inclusive, together with other material from the hospital records and other sources. The writer attempts to draw conclusions from those cases that were radiographed and/or treated in the X-ray Department of the hospital during the twelve-month period that x-ray therapy was used in the treatment of acute pneumonia. For the purposes of this paper, only those pneumonia cases that were treated and/or radiographed are analyzed extensively, but there were others that were not radiographed, for one reason or another. These also are included in several of the tables, in order to be as accurate as possible and to secure a control group.

To quote from a personal letter from Alexander D. Langmuir, M.D., Medical Consultant of the Bureau of Pneumonia Control, of the Department of Health of the State of New York, under date of Dec. 15, 1938: "Reliable statistics to make fair comparisons are very difficult to obtain, because of the tremendous number of variables which are always operating."

It is with full cognizance of the truth of this statement that this report is offered. It is hoped that the material herein contained will not engender, in those who may happen to have it come to their attention, that unwarranted enthusiasm which too often is accorded a somewhat new development in medical fields. Also, it should not be passed over too lightly, as being overrated, by those who are "sold" on other methods of treatment. Until sufficient data by others working on the subject may be presented to the medical profession, confirming or negating the apparent results secured to date, it would seem to us that there is a definite field for this use of x-ray therapy.

In attempting this evaluation of the use of x-ray therapy in pneumonia, it was felt that not only was the total death rate of the patients of interest, but that also the cost of the disease as indicated by the length

[1] This is one of a series of papers contributed by friends and former pupils of I. Seth Hirsch, M.D.

of hospitalization, etc., was of importance. Our records, we believe, bring out figures to show that the use of x-ray therapy in acute pneumonia has a very definite influence in decreasing the cost of pneumonia cases. In any disease, any method of therapy which reduces the death rate, the length of hospitalization, and the cost to the patient deserves consideration by physicians in general.

"Pneumonia is essentially a disease of winter and early spring. The four months from January through April include about 75 per cent of the cases in any normal year.

"In New York State, pneumonia in its various forms causes a greater loss of life than any other communicable disease and is exceeded as a cause of death only by heart disease and cancer. While these two latter are diseases, for the most part, of late life, pneumonia takes about 50 per cent of its toll during the ages of greatest usefulness. The annual loss of life from this cause in New York State is about 12,000" (3). (In the adjacent Province of Ontario there are about 2,500 deaths per year.)

Desjardins (5) says: "The death rate gives us at least an approximation of the incidence of the disease, since the case fatality rate, in lobar pneumonia at least, is quite constantly 25 per cent. Thus the pneumonia incidence rate is about four times the death rate." Therefore, we may justly estimate that in New York and Ontario there may be approximately 58,000 cases of pneumonia each year. If we estimate the cost of a case of pneumonia—taken care of outside the hospital—as only fifty dollars, and many cases will far exceed that figure, we have nearly three million dollars, probably much more, as the cost of pneumonia per year in the State of New York and the Province of Ontario. When we consider that the cost of pneumonia serum alone, where it is not supplied by the state or province, will be around one hundred dollars per case, and then also consider the loss of working time of those who are sick, the added expense to the families, etc., a figure of ten million dollars, or more, may more accurately give the picture of the financial side of the pneumonia problem. As many cases require hospitalization, special nursing, etc., for an extended period of time, due to complications, we believe that our figures may prove of interest. Of course, not all cases require hospitalization, and, as already stated, reliable statistics are difficult to obtain, therefore, the above figures may require revision either upward or downward.

Quoting from Powell (1): "The *modus*

TABLE I.—GENERAL CLASSIFICATION

Bronchopneumonia

	Under 10 yrs.		10–20 yrs.		20–30 yrs.		30–40 yrs.		40–50 yrs.		50–60 yrs.		60–70 yrs.		Over 70 yrs.	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
Rec.	2	3		1							2	1		1	3	1
Died	15	22	3		2	3		1	1	1	1	2	1			

Lobar Pneumonia

	Under 10 yrs.		10–20 yrs.		20–30 yrs.		30–40 yrs.		40–50 yrs.		50–60 yrs.		60–70 yrs.		Over 70 yrs.	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
Rec.	22	11	7	4	6	1	7	1	4	4	5	1		1	1	1
Died	4		1		1	3	1	1	4	1	4	3	4	2	1	

Shows general classification of the 172 cases of pneumonia admitted to the hospital from Oct. 1, 1937, to Sept. 30, 1938. The cases are divided into broncho- and lobar, and the Table also shows recoveries and deaths, by decades, for males and females.

operandi of radiation therapy in this and other acute infections is still unexplained. There seems to be definitely some relation between the destruction of the infiltrating leukocytes and the resolution of the inflammatory condition. It may be an increased permeability of the tissues and perhaps of the infecting organisms themselves to the natural lysins or ferments. The prompt response of the Type III cases suggests that either the capsule is dissolved or at least made more permeable to the action of the immune substances."

Smillie (4) says: "The rate at which the varieties of leukocytes mentioned are destroyed by irradiation under experimental conditions corresponds closely to the rate at which acute inflammations subside after exposure to a suitable dose of roentgen rays or radium." And further, "As a result of the disintegration of infiltrating leukocytes the antibodies, ferments, and other protective substances which these cells contained are liberated into the surrounding spaces where they become mixed with the tissue fluids and become available for defensive purposes." Results from the x-ray treatment of acute inflammatory processes seem to prove that the response to irradiation is in proportion to the degree of leukocytic infiltration.

The aforestated opinions seem to be the most reasonable explanations of the results secured by the use of x-ray therapy in acute infections that the writer has noted in the literature.

In this study of pneumonia, no case has been refused x-ray therapy when it was requested by the attending physician, even though the writer considered it as useless. The sooner x-ray therapy was used after diagnosis, the better have been the results, as reported by others, and the same is true in this series. In this series, treatments have been given to patients who were practically moribund at the time, just because it was requested. Whenever possible, the patient underwent his x-ray treatment immediately after admission to the hospital, and before being taken to his room.

The films in many of these cases are not as brilliant as might be desired. It must be remembered that all of these patients were acutely ill, and in order to disturb the patient as little as possible, only his outer garments were removed. Then a cassette was slipped under his back and a film made, usually at 36-inch target-film distance, 30 ma., and one-tenth second exposure, varying the kilovoltage to suit the thickness of the patient. After development and study of the film, the patient was taken to the therapy set-up in the next room and, without disturbing him at all, was given x-ray therapy. The factors used in this series were: 75 kv., 3 mm. aluminum filter, 200 r units, using an open lead glass bowl type of tube holder with a 15 cm. opening, cen-

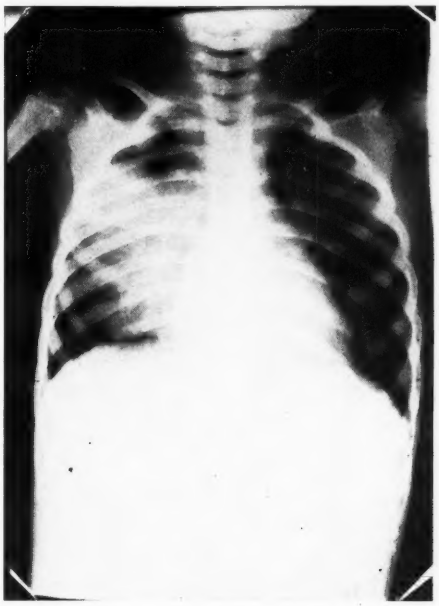


Fig. 1. Case 1. Roentgenogram of F. V. L., female, aged three, lobar pneumonia. Film made Sept. 25, 1937, four days after entering hospital, about ten days after onset of illness. Greater portion of right middle lung-field consolidated; no specific type of sputum demonstrated. X-ray therapy with bedside unit began day after this film was made. (See Chart 1.)

tering over the area of chief consolidation but without using a cone or other method of screening off the rest of the chest. In bronchopneumonia, the therapy was usually centered over the area which showed most confluence of mottling, but without excluding the entire chest. The kilovoltage used in our series has not been as high as that suggested by Powell, who, at the last reports, was using 140 kv. As the cases treated were handled entirely on an experimental basis, and in most cases only a single anteroposterior film in the supine position was made, rather than a

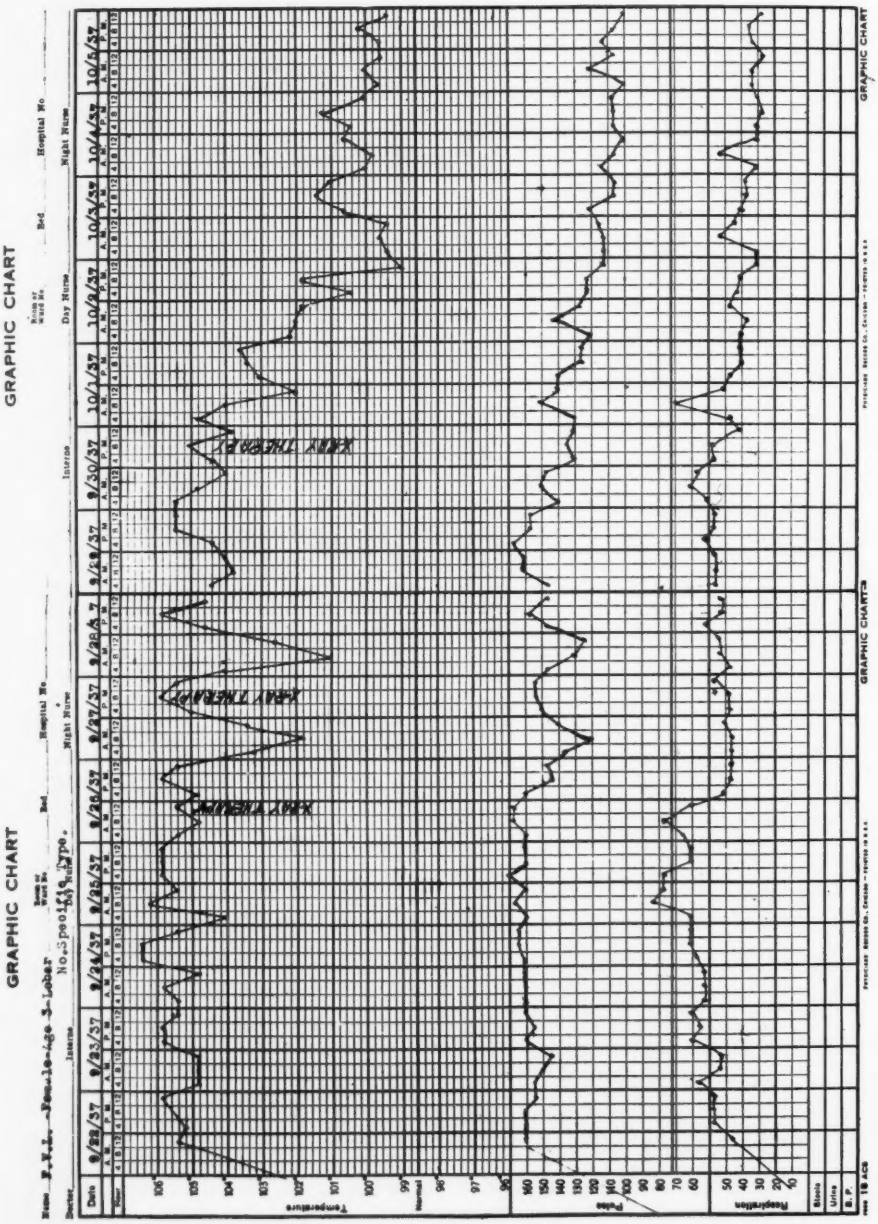


Chart 1. Case 1. Graphic chart of F. V. L. Note that respirations dropped from 75 to 45 per minute after first x-ray treatment and did not go up again; the pulse rate dropped at the same time as the temperature. Note the immediate drop in temperature after each x-ray treatment.

TABLE II.—CONSOLIDATED GENERAL CLASSIFICATION OF 172 CASES OF PNEUMONIA

Total admitted to hospital from Oct. 1, 1937, to Sept. 30, 1938

	No. Cases	M.	F.	Deaths M.	Deaths F.
Broncho-	66	30	36	7	5
Lobar	106	72	34	20	10
Total	172	102	70	27	15

Pneumonia death rate for same period

	Percentage
Pneumonia cases admitted—broncho- and lobar (172 cases—42 deaths)	23.8
Av. broncho- death rate (66 cases—12 deaths)	18.1
Av. male broncho- death rate (30 cases—7 deaths)	23.3
Av. female broncho- death rate (36 cases—5 deaths)	13.9
Av. lobar death rate (106 cases—30 deaths)	28.3
Av. male lobar death rate (72 cases—20 deaths)	27.7
Av. female lobar death rate (34 cases—10 deaths)	29.4
Av. pneumonia death rate—1935	30.8
Av. pneumonia death rate—1936	26.7
Av. pneumonia death rate—1937	32.1

TABLE III.—CONSOLIDATED GENERAL CLASSIFICATION OF 138 CASES REVIEWED

(This table includes only those cases radiographed and/or treated)

	No. Cases	M.	F.	Deaths M.	Deaths F.
Broncho-	53	27	26	5	3
Lobar	85	59	26	12	7
Total	138	86	52	17	10

	Percentage
Av. pneumonia death rate for 138 cases reviewed—broncho- and lobar (138 cases—27 deaths)	19.5
Av. broncho- death rate (53 cases—8 deaths)	15.0
Av. male broncho- death rate (27 cases—5 deaths)	18.5
Av. female broncho- death rate (26 cases—3 deaths)	11.1
Av. lobar death rate (85 cases—19 deaths)	22.3
Av. male lobar death rate (59 cases—12 deaths)	20.0
Av. female lobar death rate (26 cases—7 deaths)	23.0

complete chest study, the location of consolidation was usually reported as extending between certain levels, rather than being described as involving any special lobe. It is an interesting point, perhaps, to know what lobe is involved, but there may be some question as to actually how valuable such information is to the attending physician after he gets it. If an abscess or other cause should develop later, further studies are made when, and if, necessary. It is felt by the writer that excessive handling by the physician in making his clinical examinations in some cases of pneumonia may do more harm than good, and, likewise, it would seem undesirable to disturb the patient in the x-ray department more than is absolutely necessary.

Sante (6) says: "Lobar pneumonia may be defined as an acute infectious respiratory disease characterized by dense consolidation of one or more lobes of the lung, presenting constitutional symptoms. It is caused by the pneumococcus (in about 96 per cent of the cases) which gains entrance to the lungs by way of the trachea and bronchi and spreads rapidly toward the periphery until it finally involves the entire area of the lung supplied by the bronchiole, through which the invasion originally occurred. Occasionally, however, especially in children, it would seem that the consolidation occurs first in the periphery of the lung. In the stage of hilum consolidation within the first 24 hours of the disease, lobar pneumonia cannot be differentiated roentgenographically from acute respiratory infections such as hilum pneumonia, primary acute lung abscess, and beginning caseous tuberculous pneumonia. Within 24 hours, however, lobar pneumonic consolidation . . ." usually spreads to involve an entire lobe, thus helping to differentiate it radiographically. Lobar pneumonia usually gives a history of an acute onset.

Quoting Karsner (7): "The disease rapidly reaches its acme or fastigium, which remains for a period usually of from

seven to eleven days, to be followed in half the cases by crisis, and in the other half by lysis; convalescence may be interrupted by a variety of complications."

Bronchopneumonia (or atypical pneumonia) is a pneumonic process characterized by multiple small areas of infiltration clustering about the bronchi, which follows direct extension through the bronchial wall of infection previously present in the bronchi (6). Thus it usually has a longer history of acute respiratory infection than is the case with lobar pneumonia. No specific organism has been found to be the cause of bronchopneumonia. The roentgenogram usually shows a number of soft infiltrations with a feathery border located about the bronchi of the lower lobe. The condition is almost always bilateral and usually only in the lower lobes. The patches may be irregularly distributed, usually poorly defined, varying in diameter from 5 to 15 mm. or larger. In bronchopneumonia, "The period of fastigium or acme is of irregular duration, sometimes being relatively short, and sometimes extremely prolonged. Crisis is unusual—most of the cases tending toward convalescence after a variable period of lysis" (7).

We also have another type of pneumonia not so frequently mentioned and often considered as one stage of lobar pneumonia, that is, hilum pneumonia. It probably is an atypical type of lobar pneumonia, as the clinical symptoms and course of the disease are very similar. The physical signs of true lobar pneumonia are, of course, lacking, because the consolidation remains confined to the hilus region. The recovery is usually by crisis.

In the present discussion, the cases have been divided into two types, broncho- and lobar, the hilum type being considered as lobar.

One hundred and thirty-eight charts of the Niagara Falls Memorial Hospital, out of the 172 pneumonia cases admitted, have been reviewed and the findings divided into 50 headings and numerous sub-headings. Those studied in detail include all the pneumonia cases passing through the X-ray Department, during the period under discussion. Needless to say, not all the headings and sub-headings can be included in this presentation, nor can all the findings be discussed.

The ages of these patients varied from three weeks to 76 years. (See Table I.)

The general classification, with special attention to death rates of the various types of pneumonia, during the period under discussion, is consolidated in Table II. Also in Table II are included death rates for the years 1935, 1936, 1937, which are seen to average nearly 30 per cent. Of course, in 1935, 1936, and 1937, many patients were radiographed, but none received x-ray therapy until the last three months of 1937. Study of the 138 cases treated and/or radiographed is consolidated into Table III.

It is interesting to note, comparing Table II with Table III, that the cases which passed through the X-ray Department had approximately a 4 per cent lower death rate than the general hospital pneumonia death rate for the 12 months under consideration, but the striking point in this tabulation is the very marked difference between the patients who were given x-ray therapy and/or radiographed for diagnosis, and those who did not visit the X-ray Department at all; that is, the difference between 19.5 and 44.1 per cent. This is shown in Table IV.

Eighty-six per cent of all the pneumonia patients in the hospital during the period of this study were at least radiographed, only 34 cases not being exposed to x-ray in any way. It must be admitted that it is perhaps unfair to compare figures on a group of 34 cases with the figures on 138 cases, but this is the only means we had for securing a control group figure, so these figures are offered for what they are worth. However, as the death rate in pneumonia has often been stated to run from 30 to 50 per cent, perhaps a figure of 44.1 per cent may not be so far out of line. It almost seems impossible that the use of x-ray

(in some cases only as a diagnostic agent) could account for this tremendous improvement in death rate. However, it is not so long ago that Granger startled us with his results in the treatment of otitis media and early mastoiditis, using dosages only equivalent to the amount of x-ray used in making two films. Then again, Powell (1) remarks on improvement in pneumonia cases as a result of fluoroscopy.

In passing, it is interesting to note that in only 14 per cent of all the pneumonia cases admitted to the hospital was the attending physician satisfied to depend largely on his clinical findings, without radiographs, for evidence on which to base his diagnosis and treatment, and it is

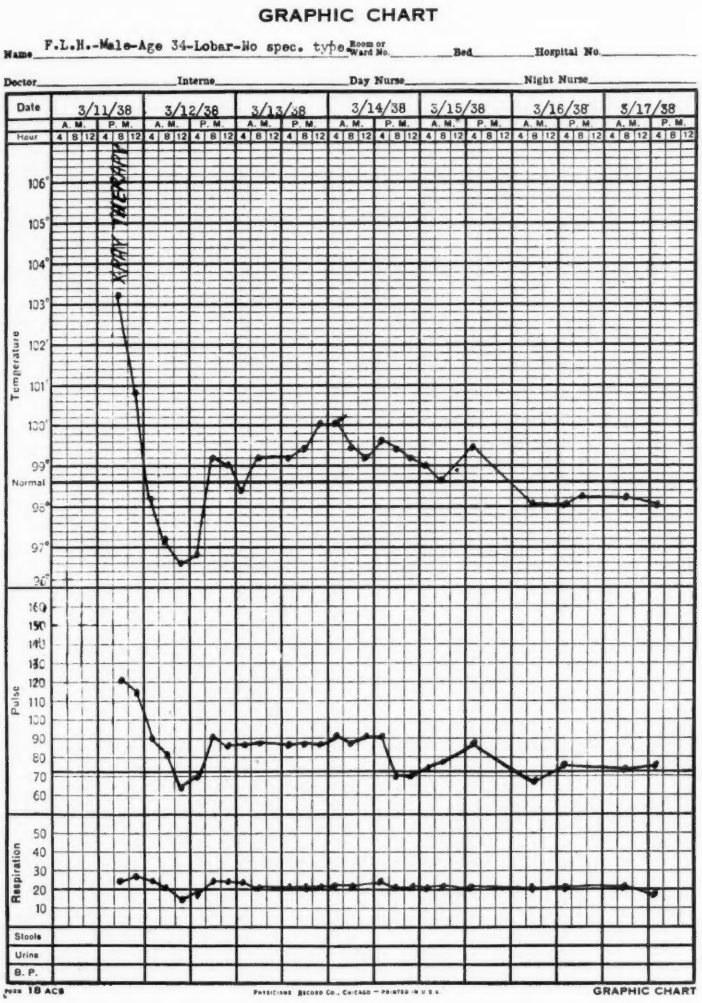
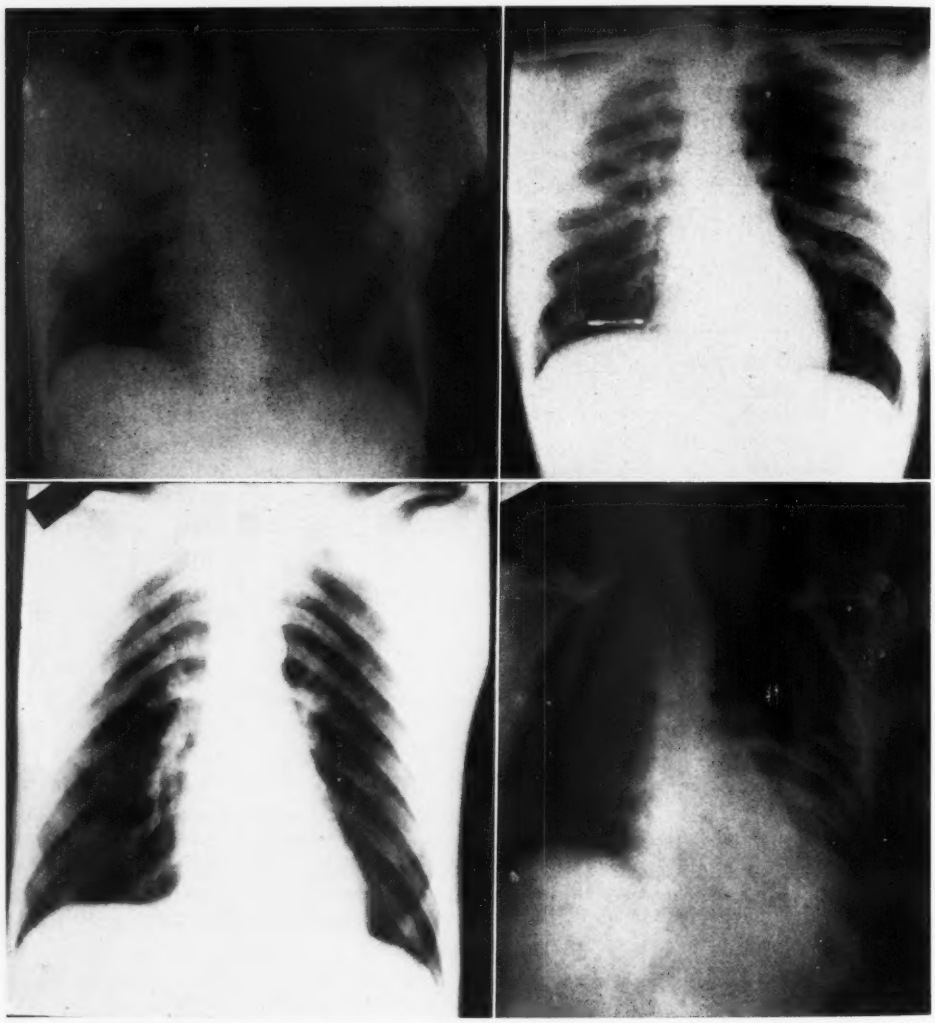


Chart 2. Case 2. Graphic chart of F. L. H. Note that temperature dropped six degrees within 24 hours after x-ray treatment was instituted; pulse and respiration rates corresponded to fall in temperature. (See Figures 2-*A* and 2-*B*, for radiographs of chest.)

in this 14 per cent that we show the 44.1 per cent death rate.

One of the first pneumonic cases that came to my attention for x-ray therapy was a patient with upper right lobar consolidation. Figure 1 shows the chest of this patient on the day before x-ray therapy was commenced. This patient was a white female, three years of age, with good family history and excellent status. At



Figs. 2-*A* and *B* (*upper*). Case 2. Roentgenogram of F. L. H., male, aged 34, lobar pneumonia, no specific type. Figure 2-*A* was made and x-ray treatment given on admission to hospital on March 11, 1938. Fig. 2-*B* was made on March 17, 1938. The patient was discharged on eighth day after admission to hospital. (See Chart 2.)

Fig. 3 (*lower left*). Case 3. Roentgenogram of H. T., male, aged 25, broncho(?)pneumonia, no specific type. Film made and treatment given on admission to hospital. No definite consolidation; only a little fine mottling present. See Chart 3, which would suggest prompt response of early acute chest infection, before stage of consolidation was reached.

Fig. 4 (*lower right*). Case 4. Roentgenogram of W. H. H., male, aged 24, lobar pneumonia, no specific type. Consolidation of lower half of left lung-field. Film made and x-ray therapy given on admission to hospital, on Feb. 22, 1938. (See Chart 4.)

the time I saw the child, she had been in the hospital for four days with a temperature of from 105 to 106°, and gave the history of having been similarly ill for at least a week at home. A small dose of x-ray therapy was given, with the bedside unit, on Sept. 26, 1937, late in the morning. Before noon the next day her temperature had dropped approximately four degrees; pulse and respiration rates also showed corresponding improvement. However, the temperature again went up nearly to 106° that afternoon, and, at that time, another slightly longer treatment was given. This time the temperature made a somewhat lower drop, and then went up again nearly to 106°. Two days were allowed to elapse without treatment, as the patient appeared generally better, and then, following a third and still heavier treatment, the patient recovered by lysis. The graphic chart of this case is reproduced in Chart 1. The apparently definite response in this case encouraged me to attempt further experiments, and the work which I am now attempting to present is the result of those experiments.

We have noted that patients frequently look better and feel better within a short time after the treatment. It frequently happens that a definite fall in temperature occurs in from 12 to 24 hours after treatment, subsequently followed by a rise, or occasionally by a rise showing two peaks, but then the temperature may proceed rather rapidly to normal. On the other hand, if the patient appears better, whether the temperature shows an abrupt drop or not, his clinical appearance is considered rather than temperature record and he is not treated for two or three days unless there seems to be need for it. Often by the time these two or three days have passed, we find that the temperature chart is slanting downward and no further treatment is necessary.

TABLE IV

	Admitted	X-rayed	Not x-rayed
Broncho-	66	53	13
Lobar	106	85	21
Total	172	138	34
Deaths	42	27	15
Death rate	24.4%	19.5%	44.1%

Demonstrates the general death rate for the 172 cases of pneumonia admitted to the hospital, as compared to those x-rayed and/or treated, and those not even radiographed.

TABLE V.—NUMBER OF TREATMENTS GIVEN PER CASE TREATED

Total no. cases treated	88
Total no. x-ray treatments given	94
Av. no. x-ray treatments given each case treated	1+
No. cases receiving x-ray therapy as chief method of treatment	63
No. treatments given cases which received chiefly x-ray therapy	67
Av. no. treatments—x-ray chiefly	1+

In our particular series the average number of treatments per case was 1+ (see Table V), and there have been only one or two cases in which x-ray therapy was properly administered early in the course of the disease that it was felt a second treatment was needed; and only in a case showing delayed resolution was a third treatment needed.

The rapid improvement in the condition of most patients who had x-ray therapy early, was, in many cases, very agreeably startling. Some patients experienced a feeling of improvement before the temperature chart demonstrated any marked improvement, while others showed simultaneous improvement with the fall in temperature.

As a matter of interest, Table VI, which gives by age groups some indication of the treatment accorded the 138 patients, is included. (Table VI is consolidated in Table VII.)

Averaging the 88 cases (Table VII), we find the average death rate for all cases having any type of x-ray treatment, no matter what else was done, to be 19.3 per cent. This does not take into con-

sideration anything as regards the method of x-ray treatment (whether by bedside unit or not), seriousness of the disease, or any other variables. This figure is so close to the figure (20 per cent) secured the results secured by simple radiography alone. Study of Table VIII, wherein each of the 27 deaths in the series has been analyzed, seems to offer some explanation of the death rate. When we remember

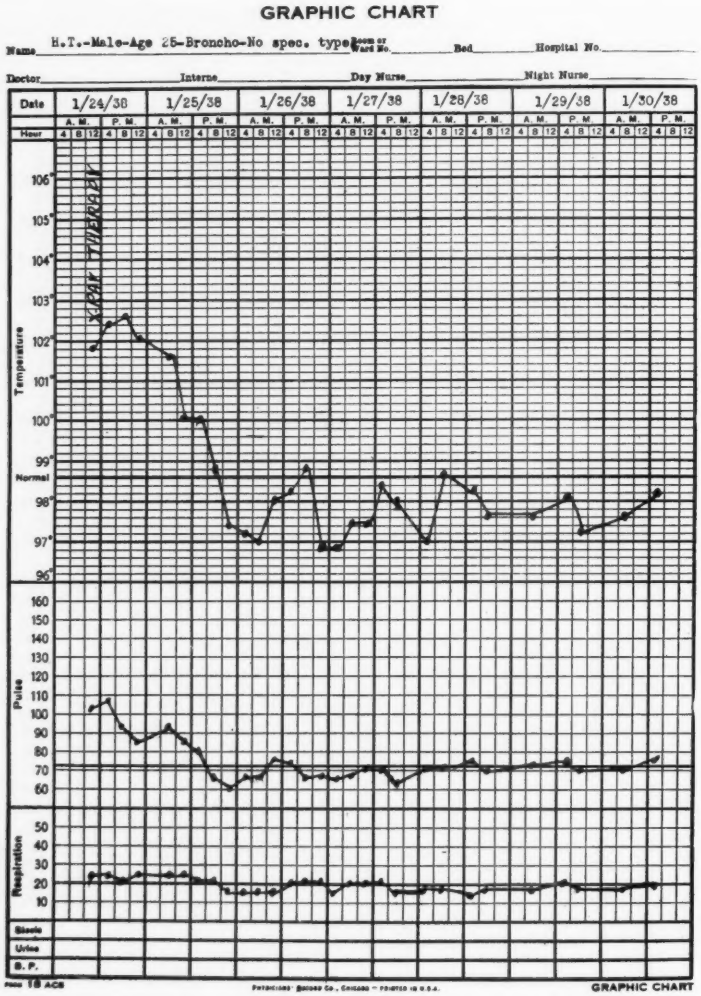


Chart 3. Case 3. Graphic chart of case H. T. (see Figure 3). Clinically considered as a bronchopneumonia on admission. Note five-degree temperature drop within 36 hours after institution of x-ray therapy. The patient was discharged in eight days. (See Figure 3 for radiograph of chest.)

for the untreated, but radiographed cases, that, on the surface, it would suggest that x-ray therapy in our series has proven of little value in reducing the mortality over the death rate for the 34 cases not even radiographed (see Table IV) to be 44.1 per cent, the death rate for our entire 138 cases is quite satisfactory.

Further analysis of Table VIII leads the writer to state definitely that only two out of the 27 fatal cases could have been classified as average pneumonias.

One of these was a lobar, Type 8, female, aged 64, who had both x-ray therapy and serum, but who did not appear to react favorably to either. She seemed to have no resistance and died within four days of entering the hospital, which was within six days of the onset of the disease. The other so-called "average" case was a lobar, Type 1, male, aged 50, suffering from a toxic myocarditis on admission, and who was "treated" with the bedside unit because he was too ill on the day after his

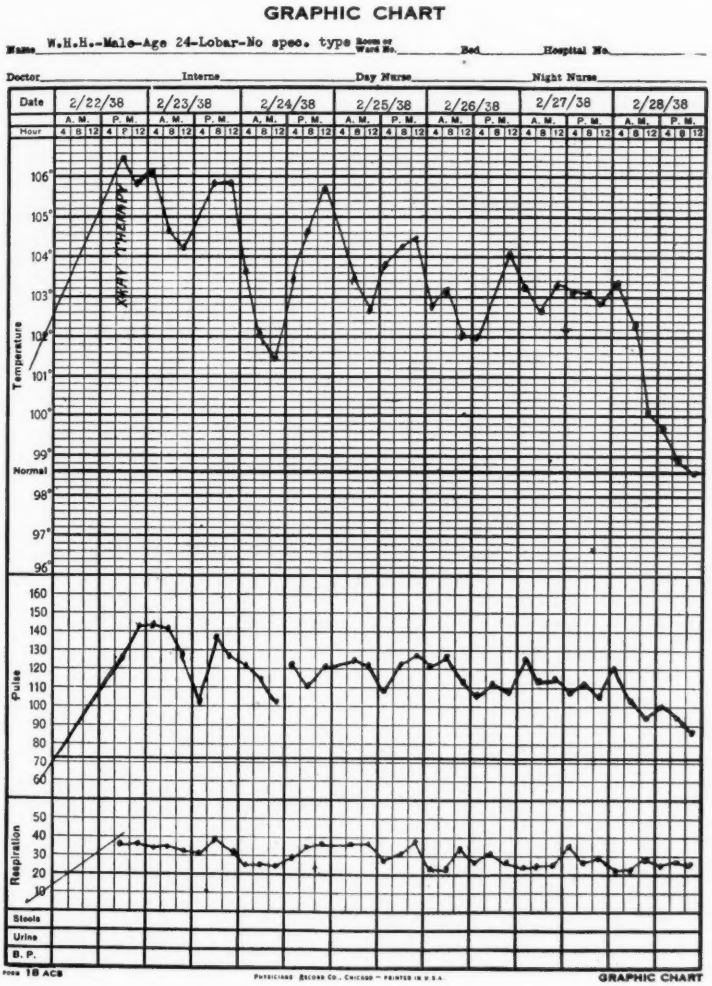


Chart 4. Case 4. Graphic chart of W. H. H. Note that temperature dropped five degrees within 36 hours of institution of the treatment. The temperature rose again, but the patient looked and felt much better than would be suggested by the chart, and further x-ray therapy did not seem necessary. He remained in the hospital 19 days. (Radiograph of the chest of this patient is shown in Figure 4.)

admission to be brought to the X-ray Department. In view of his condition when he was treated, he perhaps might better be classed as "bad" rather than "average." Our classification in this respect undoubtedly is weak, as we listed the cases according to the way they impressed us at the time of making the radiographs.

All of the other 25 fatal cases were either listed as bad or moribund, at the time of admission to the hospital. For this reason we shall not attempt to draw

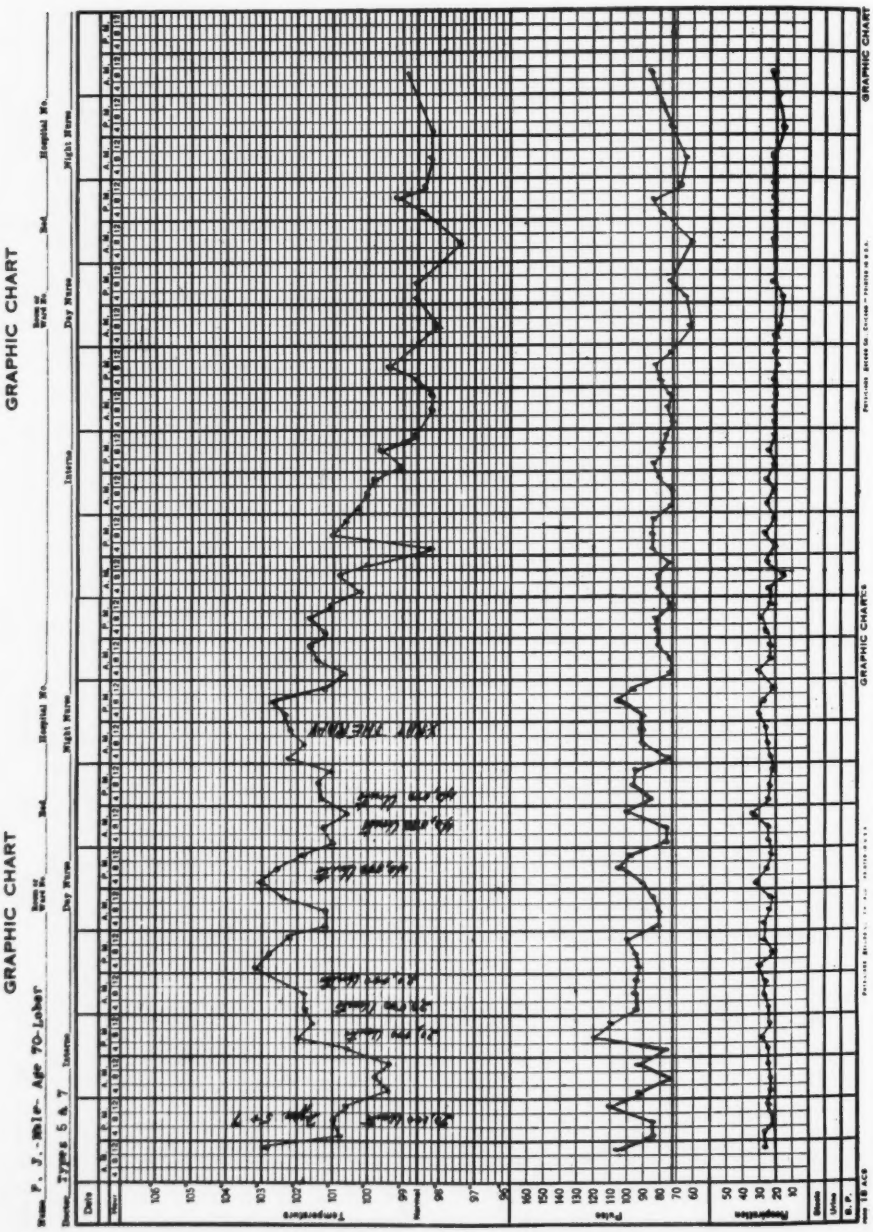


Chart 5. Case 5. Graphic chart of F. C. J. Note that patient recovered by lysis, beginning immediately after x-ray therapy. Two hundred thousand units of serum were given before x-ray therapy was instituted. A second sputum typing just before administration of x-ray therapy demonstrated a different type from that first found, and it was thought unwise to give more serum and x-ray therapy was tried. (See Figure 5 for radiograph of chest.)

any further comparisons between those who were given x-ray therapy, and those who were radiographed without having x-ray therapy, as this has already been covered in Table VII.

Of the 27 cases that died, 17 had had the essayist felt that not only was the death rate of interest in pneumonia, but that the cost of the disease as represented by length of hospitalization, etc., deserved to be considered.

We have charted, therefore, in Tables

TABLE VI.—CLASSIFICATION OF PNEUMONIAS ACCORDING TO AGE AND TREATMENT

(138 Cases)

1937–1938 Treatment		Under 10		10–20		20–30		30–40		40–50		50–60		60–70		Over 70	
		M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
X-ray therapy	Rec.	12	12	8	2	3	2	5	1	1	4	1			2		
	Died	2	2							1				2	1	2	
No x-ray therapy	Rec.	15	7	5	1	5	3	2		1		1					
	Died	1		1	1					1	1		1	3			1
X-ray and other spec. medication	Rec.	7	3		3					1	1	1				1	1
	Died	1					1			1		1		1	2		
Total	Rec.	34	22	13	6	8	5	7	1	3	5	3			2	1	1
	Died	4	2	1	1		1			3	1	1	1	6	3	2	1
Moribund at time of giving x-ray (c) complications (p) portable therapy		2(c) 1(p)	2(c)				1(p)			1(p)				2 (1c)	1 (1c)	1 (1p)	

Showing in general, by age groups, the number of cases that recovered, and the number of cases that died, giving indication of the treatment given these cases. This Table includes only the 138 cases which passed through the X-ray Department for x-ray therapy and/or radiographs. The cases are herein considered under the general heading of pneumonia and not divided into broncho- and lobar variations.

x-ray treatment. Although most of these 17 were treated immediately upon entrance to the hospital, and, therefore, they are listed as having been treated "early," nevertheless many of them were manifestly treated "too late" to offer much hope of aiding the recovery of the patient.

Treatment of a patient considered as moribund on admission to the hospital, even though the patient was treated as soon as he or she was admitted, could hardly be expected to turn the course of the disease. Incidentally, six of the 17 fatal cases were "treated" with the bedside unit because the attending physician felt that they were too ill to be moved to the X-ray Department. We have not felt that cases treated with the bedside unit should be chalked up as failure of the use of x-ray treatment, as the bedside unit is of low capacity and not designed for therapy.

As stated earlier in this presentation,

TABLE VII.—CONSOLIDATION OF TABLE VI

	No. cases	Outcome	Males	Females	Death rate M.	Death rate F.	Average death rate
Chiefly x-ray therapy	63	Recovered	30	23	18.9%	11%	15.8%
		Died	7	3			
X-ray and other medication	25	Recovered	10	8	23%	33.3%	28%
		Died	3	4			
No x-ray therapy	50	Recovered	28	12	17.6%	25%	20%
		Died	6	4			

Demonstrates the death rates of the 138 cases. It is interesting to average the deaths of all those patients who had x-ray therapy, no matter what else was done, and secure the figure 19.3 per cent which is very close to the 20 per cent figure for those patients radiographed, but not given any x-ray therapy.

IX and X, the average length of time the patients were found to have remained in the hospital, with and without x-ray therapy, and with various combinations of

treatments, dividing the cases into age groups.

Summarizing Tables IX and X in a more general way, we submit Table XI.

It appears that when we consider the

Fig. 5. Case 5. Roentgenogram of F. C. J., male, aged 70, lobar pneumonia. First sputum examination showed Types V and VII. Illustration shows appearance of lung-fields on Jan. 8, 1938, the day the x-ray treatment was given. (See Chart 5.)

138 reported cases, that the average hospitalization time for a pneumonia case in our series, whether the patient recovers or not, and considering all types of treatment is: Males, 15.9 days; females, 13.9 days.

Study of Table XI leads us to the opinion that the length of time taken by a patient in the hospital to recover, who is treated chiefly by x-ray therapy, may be expected to be quite definitely shorter than the time required when treated by x-ray combined with other methods, and approximately one-half the number of days as compared to the time required when the patient receives no x-ray therapy at all (although radiographed), no matter what other type of treatment may be used. If we can cut the length of hospitalization in half, we certainly cut the cost to the patient.

In order to go into somewhat more detail as to the sputum types of pneumonia that were reported to us by the clinical laboratory, and the treatment accorded these cases, and their final outcome, we refer to Table XII.

Only 60 cases, or less than half of our total series, were typed. We believe that all pneumonia cases should be typed by the laboratory as early as possible, if the use of serum is to be considered at all. In our hospital, this kind of laboratory work, for which a charge is made, is done only on request, and in many instances, if the clinical appearance of the case is satisfactory, this procedure is omitted, in order to keep down the patient's expense.

Blood cultures were done in so few cases that we are not even reporting them. As a general thing it is admitted that only 30 per cent of all blood cultures are positive for pneumococcus, and this probably accounts for the lack of enthusiasm regarding this procedure in our hospital.

For those who may be interested in our use of oxygen in this series, we refer to Tables IX and X. However, in order to summarize the use of oxygen in our cases in a more orderly manner, see Table XIII. In the Niagara Falls Memorial Hospital, oxygen is given almost entirely by the tent method. Seventy-four cases were given oxygen for varying lengths of time, as is shown in Table XIII. It is very interesting to note that in our series those patients who were treated chiefly by x-ray therapy required oxygen less than half as long as those who had no x-ray treatment and only about two-thirds as long as those who were given x-ray and other treatment.

The treatment of pneumonia by oxygen, plus 5 to 10 per cent carbon dioxide, was described by Alison (8), in 1932, as giving very satisfactory results in early pneumonias, particularly if given within the first 36 hours of the onset of the disease. However, probably in view of the excellent results reported by Evans and Durshordwe (9), of Buffalo, N. Y., with whom our staff members are personally acquainted, we, in Niagara Falls, have not used the carbon

TABLE VIII.—DEATHS

X-ray No.	Initials	Sex	Age	Broncho- or Lobar	Type	Condition on admission	Duration of disease before admittance	Total stay in hospital	Complications on admission	No. of Trts.	Trt. Early	Too Late	Portable	Serum—Yes	Serum—No	Sulph.—Yes	Sulph.—No	No. days O_2	Not given O_2	Complications during hospitalization. (Continued from admission or occurred during hospitalization: see previous column)
808-37	LB	M	15	Lob	No Sp.	Bad	6 da.	.5 da.		0					x		x	3		Otitis media—Pericarditis
736-37	AT	M	64	Lob	Not Typed	Bad	7 da.	.5 da.		1	x	x	x		x		x	5		Uremia
720-37	WH	M	3 mo.	Lob	Not Typed	Bad	?	10 da.		1		x			x		x	10		Atelectasis
721-37	DM	M	6	Lob	Not Typed	Bad	4 da.	10 da.	Appendix	1	x	x	x		x		x		x	Peritonitis
635-37	DC	M	42	Lob	No Sp.	Bad	7 da.	5 da.	Cardiac	1	x	x	x		x		x	5		Acute cardiac dilatation
613-37	JD	M	75	Bro	No Sp.	Bad	3 da.	5 da.	Cardiac	1	x	x			x		x	5		Cardiac hypertrophy
472-37	JB	F	44	Lob	Not Typed	Bad	?	11 da.	Abortion	0					x		x	4		Septicemia
432-37	LK	M	40	Lob	No Sp.	Mor	13 da.	2 da.	Nephritis	0					x		x	2		Toxemia
340-37	GW	M	63	Lob	No Sp.	Bad	7 da.	5 da.	Tuberculosis	0					x		x	3		Tuberculosis
321-37	PF	M	60	Bro	No Sp.	Bad	3 da.	5 da.	Cirr. Liver	0					x		x	5		Nephritis
154-37	JT	M	64	Bro	No Sp.	Mor	4 da.	2 da.	Heart	0					x		x	2		Dilated heart
85-37	CM	F	52	Bro	Not Typed	Bad	?	3 da.	Hernia & Hemiplegia	0					x		x	3		Old hemiplegia
72-37	LW	M	9 mo.	Bro	No Sp.	Bad	3 da.	96 da.		0					x		x	96		Empyema
70-37	LM	F	13	Lob	No Sp.	Bad	?	9 da.	Myocarditis	0					x		x	6		Toxic myocarditis
43-37	TB	F	70	Lob	No Sp.	Bad	3 da.	6 da.		0					x		x	4		
185-38	JD	F	64	Lob	8	Avg	2 da.	4 da.	Cardiac	1	x			x			x	4		Cardiac
149-38	CC	M	50	Lob	7	Bad	1 da.	4 da.	Myocarditis	1	x			x			x	4		Myocarditis
142-38	JP	M	63	Lob	Not Typed	Mor	2 da.	2 da.	Heart	1	2		x		x		x		x	Heart
129-38	JL	M	76	Bro	Not Typed	Bad	10 da.	5 da.	Myocarditis	1	x				x		x	4		Myocarditis—ascites
87-38	GW	M	48	Lob	1	Bad	5 da.	6 da.		2	x			x			x	8		Pleurisy—Abscess of Lung
54-38	DT	M	50	Lob	1	Avg	6 da.	3 da.	Myocarditis	1	x		x	x			x	3		Toxic myocarditis
221-38	JT	F	63	Lob	3	Bad	6 hr.	4 da.	Tuberculosis	1	x			x			x	4		Sup. of urine
395-38	RC	F	24	Lob	No Sp.	Bad	?	12 da.	Exoph. Goiter	1		x	x		x		x	6		Goiter—Surg.
375-38	GL	F	59	Lob	1	Mor	2 da.	3 da.		1	x				x		x	3		Hypertension—Nephritis
321-38	GP	M	64	Lob	5	Mor	9 da.	3 da.		1	x	x			x		x	3		Pleurisy
536-38	HM	F	1	Bro	Not Typed	Bad	5 da.	3 da.	Enl. Heart	1	x	x			x		x	3		Enlarged heart
472-38	BB	F	17 mo.	Bro	No Sp.	Mor	?	2 da.	Very Toxic	1	x				x		x	2		Severe toxemia

This is an analysis of the 27 fatal cases in the series of 138 given x-ray therapy and/or radiographed. Note that all but two were considered as "bad" or "moribund" at the time of admission to the hospital.

Note: "No Sp." type means that no specific type was found although types one through eight were searched for.

TABLE IX.—LOBAR PNEUMONIA

Total Number of Days Spent in Hospital

(Classification According to Age and Treatment)

Age	Under 10				10–20				20–30				30–40				40–50				50–60				60–70				Over 70			
	Rec.		Died		Rec.		Died		Rec.		Died		Rec.		Died		Rec.		Died		Rec.		Died		Rec.		Died		Rec.		Died	
	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
Chiefly x-ray plus O_2	43	65	10		31					14		12	22					9	5				3			36	8					
No. cases	4	4	1		3					1		1	2					1	1				1			1	1					
av. days	11	16	10		10					14		12	11					9	5				3			36	8					
No O_2 (had x-ray)	35		10		22	9			19				31	41			18	38			30						2					
No. cases no O_2	3		1		3	1			1				3	1			1	2			1						1					
av. days	12		10		7	9			19				10	41			18	19			30						2					
X-ray plus serum and sulph. and O_2		30																			18					69						
No. cases		1																			1					1						
av. days		30																			18					69						
No O_2																																
No. cases no O_2																																
av. days																																
X-ray and serum plus O_2	39																18	21	6				4				8	27				
No. cases	4																1	1	1				1				2	1				
av. days	10																18	21	6				4				4	27				
No O_2 (had x-ray and serum)	24				13																											
No. cases no O_2	2				1																											
av. days	12				13																											
X-ray and sulph. plus O_2																																
No. cases																																
av. days																																
No O_2 (had x-ray and sulph.)					13																											
No. cases no O_2					1																											
av. days					13																											
Serum and sulph. and O_2													14																			
No. cases													1																			
av. days													14																			
No O_2 (had serum and sulph.)																																
No. cases no O_2																																
av. days																																
Serum and oxygen no x-ray	31																															
No. cases	1																															
av. days	31																															
No O_2 (had serum)									18																							
No. cases no O_2									1																							
av. days									18																							
Sulph. plus O_2	52				8																											
No. cases	1				1																											
av. days	52				8																											
No O_2 (had sulph.)	18								14				12																			
No. cases no O_2	1								1				1																			
av. days	18								14				12																			
No spec. trt. but oxygen	68						5		12	12									2	11							5				6	
No. cases	3						1		1	1									1	1							1				1	
av. days	22						5		12	12									2	11							5				6	
No. spec. trt.—no oxygen	18				31				17								8				77											
No. cases no O_2	1				1				1								1				1											
av. days	18				31				17								8				77											

Number of days spent in the hospital and the length of stay with various combinations of treatments used, and recoveries and deaths according to age groups in the cases of lobar pneumonia. Also, when oxygen was or was not used with other medications. (Explanatory example—First column: Four males, under 10 years of age were treated with oxygen, but the main dependence, aside from oxygen, was placed on x-ray therapy. These four cases remained in the hospital a total of 43 days, or an average of 11 days per case.)

TABLE X.—BRONCHOPNEUMONIA

Number of Days Spent in Hospital

(Classification According to Age and Treatment)

Age	Under 10				10–20				20–30				30–40				40–50				50–60				60–70				Over 70			
	Rec.		Died		Rec.		Died		Rec.		Died		Rec.		Died		Rec.		Died		Rec.		Died		Rec.		Died		Rec.		Died	
	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
Chiefly x-ray plus O_2	49	51		50					6	14																8						10
No. cases	4	5		2					1	1																1						2
av. days	12	10		25					6	14																8						5
No O_2 (had x-ray)		21			11	9			8									24														
No. cases no O_2		3			1	1			1									1														
av. days		7			11	9			8									24														
X-ray and sulphanilamide plus O_2	18																															
No. cases	2																															
av. days	9																															
No O_2 (had x-ray and sulph.)		30			18	10																										
No. cases no O_2		1			1	1																										
av. days		30			18	10																										
Serum and x-ray plus O_2																					3											
No. cases																					1											
av. days																					3											
No O_2																																
No. cases no O_2																																
av. days																																
No specific treatment (general care), no O_2	82	87			18				34	18																						
No. cases	4	5			1				1	1																						
av. days	20	17			18				34	18																						
No specific treatment with O_2	116	29	96			26	9			23												3	7									
No. cases O_2	4	2	1			1	1			1												1	2									
av. days	29	14	96			26	9			23												3	3									

The bronchopneumonias. Similar to Table IX which covered the lobar pneumonias (see footnote, Table IX)

dioxide addition. The Alison report is of interest and there would seem to be no contra-indication for the use of this type of treatment in addition to x-ray therapy, if so desired by the attending physician.

Only 15 patients in our series were given sulphanilamide; one of these died. This group is too small to draw any conclusions, and, therefore, it is reported only as a matter of interest.

In our series, 21 patients received serum. In at least one case, after giving 200,000 units without satisfactory clinical results, another sputum examination showed a different type than at first, and it was not felt advisable to resort to more serum. X-ray therapy, at this stage of the disease, was followed by response within 12 hours, and the patient then recovered by lysis and was discharged six days later. It might appear that x-ray therapy should not be credited with the recovery of this patient, since cases which are given serum may recover by lysis (3), but the clinical improvement did follow almost immediately after the x-ray therapy and, therefore, the x-ray therapy deserves at least some consideration.

The possibility of serum reactions, of course, does not preclude giving serum to sensitive patients, unless these symptoms are of an unusually severe character (3). However, we must beware of allergic patients, or those who give a history of asthma, hay fever, eczema, etc. Also, we may expect serum reactions if tetanus serum has been given within six months. If no serum of any kind has been given

within two years, however, and the history is clear, there probably will be little or no reaction. As every physician knows, serum reactions may occur at any time following the administration of the serum—from an immediate reaction up to ten days or later—and be of all stages of severity from sudden death to an urticaria. With the use of x-ray therapy, in the moderate amounts which seem to affect acute pneumonias, we do not have to consider reactions. Although very enthusiastic over our results with this mode of treatment, we hardly yet feel justified in withholding serum when the sputum typing indicates that a specific serum is available. Table XIV lists the serum reactions (of any degree) that occurred in our series (42.8 per cent).

Powell (1) stated, "Complications still occur with about the expected frequency." (In our series there were eight patients who received x-ray who had complications—out of 87 treated—or approximately 9 per cent.) This compares with 12 patients out of 41 (29 per cent) who did not have x-ray therapy but who did have complications. This would seem to be too large an improvement in favor of the x-ray treated cases to be lightly overlooked.

Careful review of the cases with the attending physicians, leads us to feel that in the series reported the satisfactory outcome was due to x-ray therapy in not less than 54 cases (probably many more), but, in arriving at that figure, all cases in which there seemed to be any possible room for argument were eliminated.

TABLE XI.—SUMMARY OF TABLES IX AND X

Number of Days Spent in Hospital

			M.			F.		
			Chiefly X-ray	X-ray and Other	No X-ray	Chiefly X-ray	X-ray and Other	No X-ray
Broncho-	Recovered	No. patients	7	3	12	12	1	10
		No. days	74	36	300	127	20	182
		Av. no. days	10	12	25	10	20	18
	Died	No. patients	2	1	2	2	..	1
		No. days	10	3	72	50	..	3
		Av. no. days	5	3	36	25	..	3
Lobar	Recovered	No. patients	21	9	17	12	6	1
		No. days	251	126	398	228	175	12
		Av. no. days	12	14	23	19	29	12
	Died	No. patients	6	3	3	2	2	3
		No. days	43	16	11	15	8	22
		Av. no. days	7	5	3	7	4	7

Patients treated chiefly by x-ray remained in the hospital about half as long as those who had no x-ray therapy.

TABLE XII.—CLASSIFICATION OF PNEUMONIAS ACCORDING TO TYPE

Treatment	Type I		Type II		Type III		Type IV		Type V		Type VII		Type VIII		Non-spec.	
X-ray only	Rec. 1	Died 1	Rec. 1	Died	Rec.	Died	Rec. 1	Died	Rec.	Died 1	Rec.	Died	Rec. 2	Died	Rec. 23	Died 4
X-ray and other	9	2			2	1	2					1		1	6	
Serum only, no x-ray	2															

Note: "Non-spec." means that no specific type was found although types one through eight were searched for.

SUMMARY AND CONCLUSIONS

1. A series of 138 cases of pneumonia have been reviewed (of which 88 had some form of x-ray therapy) in an attempt to demonstrate the value of x-ray therapy in the treatment of acute pneumonia.

2. In x-ray therapy we have a simple and easy method of treatment which appears to influence favorably the course of many cases of acute pneumonia. Equipment for giving this type of treatment is now available in most hospitals; but it should be supervised by a qualified roentgenologist and given with properly calibrated equipment.

3. The length of hospitalization is decreased by x-ray therapy.

4. The temperature returns to normal more quickly.

5. The frequency of complications seem definitely lessened.

6. The death rate, as compared to that from other methods of treatment, has been lower, as long as the patients were even radiographed, but as compared to cases not even radiographed the percentage in favor of some form of x-ray seems almost incredible.

7. Contra-indications appear to be *nil*, whereas other types of medication, especially serum, may be definitely contra-indicated.

8. Any other treatment desired by the attending physician may be given in conjunction with x-ray therapy.

9. No reactions to x-ray treatment have been noted in this series, while serum reactions may be of varying degrees.

10. Many cases of pneumonia are hospitalized too late to expect good results from any type of therapy. Education of the public and of the medical profession is the only way to overcome this tendency to delay hospitalization. Practically all the cases that died (in this series) had a bad prognosis from the time of admission to the hospital.

11. Further reports from others working on this problem are necessary in order to estimate accurately the value of x-ray therapy in the treatment of pneumonia, as this series is somewhat small and the treatments by x-ray were too often complicated by other medication.

TABLE XIII.—USE OF OXYGEN

Totals	Chiefly with X-ray		Without X-ray		With X-ray and Other Treatment	
	No. Patients	Days	No. Patients	Days	No. Patients	Days
Lobar	21	95	11	39	14	94
Broncho-	11	49	10	155	7	36
Total	32	144	21	194	21	130
Average	$4^1/_2$ days		$9^1/_4$ days		$6^1/_5$ days	

TABLE XIV.—X-RAY REACTIONS *vs.* SERUM

Total No. Cases Reviewed	138
Total no. cases receiving x-ray (either alone or combined)	87
Total no. x-ray reactions	0
Total no. patients receiving serum	21
Total no. serum reactions	9
Percentage of serum reactions of varying degree—sufficient to be reported in daily notes of nurses or physicians	42.8%

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BIBLIOGRAPHY

(1) POWELL, EUGENE V.: Roentgen Rays in the Diagnosis and Treatment of Pneumonia. Texas State Jour. Med., **33,** 427–432, October, 1937.

(2) Idem: Roentgen Therapy of Lobar Pneumonia. Jour. Am. Med. Assn., **110,** 19–22, Jan. 1, 1938.

(3) Bureau of Pneumonia Control, New York State Department of Health. Clinical Aspects of Pneumococcus Pneumonia, 1936, pp. 5, 10, 14.

(4) SMILLIE, WILSON G.: The Prevention of Pneumonia. New York State Jour. Med., **38,** 1485–1491, Dec. 1, 1938.

(5) DESJARDINS, ARTHUR U.: The Action of Roentgen Rays or Radium on Inflammatory Processes. RADIOLOGY, **29,** 436–445, October, 1937.

(6) SANTE, L. R.: The Chest Roentgenologically Considered, **11,** chap. 15. Paul B. Hoeber, New York City, 1930.

(7) KARSNER, H. T.: Human Pathology, pp. 551 and 564. Lippincott, Philadelphia, 1926.

(8) ALISON: South. Med. Jour., April, 1932.

(9) EVANS, JOHN H., and DURSHORDWE, C. J.: Further Observations on Oxygen Therapy in the Treatment of Pneumonia. Anesth. and Analg., **11,** 193–201, September-October, 1932.

LIPIODOL GRANULOMA IN FALLOPIAN TUBES LOCALIZED BY INTRA-UTERINE DIODRAST INJECTION, WITH SPECIAL REFERENCE TO THE VALUE OF FOLLOW-UP X-RAY FILMS[1]

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LIPIODOL has been used in conjunction with the x-rays as a diagnostic test of tubal patency since the report of C. Heuser (1), in 1924. Its radiopacity was first appreciated by Sicard and Forestier (2), who recommended this iodine oil compound for general diagnostic adoption, in 1922. The literature contains numerous reports in which excellent radiographic films are reproduced showing shadows of lipiodol within the tube lumen as well as the "spill" into the peritoneal cavity.

However, in gynecology, especially in the investigation of sterility, a number of reports of the last ten years has brought to light certain undesirable sequelæ which are generally overlooked and perhaps under-emphasized.

The purpose of the present communication is to call attention to one of these sequelæ, namely, the persistent retention of lipiodol within the fallopian tubes and to the need of taking follow-up films in such cases.

In a previous paper (3) I have reported a follow-up of 43 patients who gave a history of having had an intra-uterine injection of lipiodol. In 22 of these, a skiagram of the pelvis was subsequently available. Fifteen of the 22 showed some lipiodol residue. In the same paper reference was made to Rabbiner's (4) report of contrast media in the pelvis demonstrated by an x-ray plate which was taken in four cases several weeks and months after lipiodol injection. Since then an opportunity has presented itself of studying the films in five additional instances. They were taken from an unselected group of 31 cases, in 26 of which for various reasons a film was not obtained. Lipiodol residue was present in three of the five cases for from one month to a year after the injection. Whether and to what extent deposits of lipiodol may have been encountered in the 26 cases not controlled later by an x-ray plate, is purely conjectural. Opacities found after intervals of more than a year may be so reduced in size as to be confused with phleboliths or small ureteral calculi. In a recent case lipiodol residue was found scattered in the pelvis four years after the injection.

Although the presence of post-injection opacities may not be of further significance than perhaps to suggest the presence of peritoneal cysts or adhesions surrounding the oil globules, the presence of foreign body granuloma within the tube lumen which results from persistent irritation of the retained oil is of greater clinical importance. This applies particularly in a patient who has been investigated by lipiodol for sterility and whose tubes were only partially obstructed. I have reported three such instances of tubal blockade due to lipiodol residue, in the communication mentioned above, and subsequently, met with several additional cases. These corroborate the earlier report by E. Ries (5), which dealt with foreign body reaction induced by lipiodol in the tubes. Similar reports were published by J. Novak (6), G. Albano (7), R. S. Hoffmann (8, 9), W. Odenthal (10), R. Zimmerman and H. Nahmmacher (11), R. Schröder and H. Jacobi (12), and G. K. F. Schultze (13).

In order to identify the lipiodol residue in the tubes, it is obviously necessary to introduce another x-ray opaque substance

[1] This is one of a series of papers contributed by friends and former pupils of I. Seth Hirsch, M.D.

into the uterus. However, a second injection of lipiodol is not advisable nor has this been generally adopted by ardent advocates of iodized oil. For this reason I have felt that when a radiopaque fluid is to be reinjected it is best to utilize one of the crystalloid organic iodides commonly used in intravenous urography such as hippuran, skiodan, or diodrast (Neustaedter, 14, Titus, 15, Morse and Rubin, 16). These afford excellent radiopaque shadows and owing to their rapid resorption leave no residue within the uterus, tubes, or peritoneal cavity. As the fluid passes through the tube, it is apt to be totally or partially blocked by the organized lipiodol residue. The procedure is analogous to the method employed to locate the presence of stone in the ureter by injecting such media intravenously or through the ureteral catheter.

A case recently under my observation illustrates the application of intra-uterine diodrast injection for locating the obstructed portion of the fallopian tube and of identifying the lipiodol residue in it.

Fig. 1. Fig. 2.

Fig. 1. M. McG. Lipiodol residue four months after intra-uterine injection. Several opacities appearing on the film were not identified as lipiodol residue, resembling possible phleboliths or ureteral calculi.

Fig. 2. M. McG. Diodrast injection showing the left isthmus down to but not past the irregular shadow seen in Figure 1. This shadow disappeared with the removal of the ampullary portion of the left tube which, on microscopic section, showed foreign-body giant cells resembling tubercles.

Mrs. M. McG., 29 years old, married four years, had been trying to become pregnant for the past two years. She stated that she had had a tubal insufflation which demonstrated non-patency. On June 27, 1938, a second tubal insufflation also demonstrated non-patency. She was admitted to the gynecological service of Mount Sinai Hospital on Oct. 24, 1938, for further study and operation. Preliminary to injection of diodrast into the uterus, a "flat plate" was taken which showed an irregular shadow, caterpillar-like, in the region of the lower left portion of the sacrum (Fig. 1). This proved to be the residue from a lipiodol injection which the patient recalled having had four months previously. Diodrast was then introduced into the uterus. The x-ray film showed no shadow corresponding to the right tube.

On the left side the opaque fluid was seen reaching down to the shadow found on the flat plate (Fig. 2).

At operation, Nov. 3, 1938, the uterus was found in retroflexion, bound down to the posterior cul-de-sac by numerous fine and filmy band-like and string-like adhesions. Both tubes were markedly thickened and bound down with the ovaries deeply in the cul-de-sac by numerous filmy adhesions. The right tube was the seat of nodular thickenings which were marked at its inner half. On the left side at the cornual angle just beneath the tube, a yellowish caseous mass the size of a pigeon's egg was located. In order to determine the part of the tubes which remained patent, a uterine cannula connected to an insufflation apparatus was introduced into the uterine cavity. The lumen of the right tube was thus found to be completely obliterated; hence it was removed. The left tube was found to be thickened and nodular and was removed segment by segment from the fimbriated end toward its uterine insertion, till a point 1.5 inches from the latter was reached, when CO_2 gas was seen to percolate through the lumen.

A flat plate taken after the operation showed that the residual shadow originally seen was no longer present. The portion of the left tube which contained the lipiodol residue showed, on section, caseating salpingitis *resembling* tuberculous salpingitis but no tubercle bacilli were found. Incidentally, the mass of caseous degeneration was found to consist of a cystic structure with complete fibrosis of the cyst wall, the inner surface showing caseation with calcium deposits; no ovarian tissue was recognizable.

It is noteworthy that the diodrast was completely absorbed after one-half hour. Eighteen days following the laparotomy, an insufflation demonstrated the left isthmic portion to be patent but stenosed. Diodrast passed readily through this stenosed tube end and was rapidly resorbed. There was no sign of peritoneal irritation either as a result of the repeated insufflations or of the diodrast injections.

SUMMARY AND CONCLUSIONS

Lipiodol injection into the uterus is frequently followed by retention of the iodized oil in the tubes as well as in the pelvic and upper abdominal cavity (17). Eighteen out of 27 cases in which a follow-up film was available showed opaque deposits at periods of from one month to one year and longer.

Retention is common when the tubes are sealed or partially patent. The damage is more serious in the latter cases in which the partially permeable lumen becomes obliterated by organization and foreign body granuloma. The value of follow-up films is emphasized. If a radiopaque substance is to be used, the crystalloid iodides have the advantage of being injected more than once and are especially useful for purposes of identifying lipiodol residue within the tube lumen.

REFERENCES

(1) HEUSER, C.: La Radiografia de los Ovarios, las Trompas y la Matriz, con o sin Inyeccion de Substancia Opaca o con Neumoperitoneo. Semana méd., **3,** 1496, 1924.

(2) SICARD, J. A., and FORESTIER, J.: Méthode générale d'exploration radiologique par l'huile iodée (lipiodol). Bull. et mém. Soc. d. méd. d. hôp. de Paris, **46,** 463–469, March 17, 1922.

(3) RUBIN, I. C.: Retention of Lipiodol in Fallopian Tubes, with Special Reference to Occlusive Effect in Cases of Permeable Strictures. N. Y. St. Jour. Med., **36,** 1089–1096, Aug. 1, 1936.

(4) RABBINER, B.: A Comparative Study of Lipiodol Injection and Air Insufflation in Sterility. Am. Jour. Obst. and Gynec., **29,** 100–105, January, 1935.

(5) RIES, E.: Effect of Lipiodol Injection on the Tubes. Am. Jour. Obst. and Gynec., **17,** 728, 1929.

(6) NOVAK, J.: Salpingographie oder Tubendurchblasung? Zentralbl. f. Gynäk., **54,** 3013–3019, Nov. 29, 1930; **55,** 1449, 1450, April 25, 1931.

(7) ALBANO, G.: Was geschieht mit dem eingeführten Jodöl nach einer Salpingographie? Zentralbl. f. Gynäk., **53,** 1894–1897, July 27, 1929.

(8) HOFFMANN, R. S.: Schädigung durch Salpingographie. Zentralbl. f. Gynäk., **52,** 1201–1206, May 12, 1928.

(9) Idem, R. S.: Zur Frage der Schädigung durch Salpingographie. Zentralbl. f. Gynäk., **54,** 2776–2780, Nov. 1, 1930.

(10) ODENTHAL, W.: Ueber Gefahren der Uterosalpingographie. Zentralbl. f. Gynäk., **51,** 1824, 1927.

(11) ZIMMERMAN, R., and NAHMMACHER, H.: Ist bei der Hystrosalpingographie eine lokale oder allgemeine schädigung durch das iodipin als kontrastmittel zu befürchten? Fortschr. a. d. Geb. d. Röntgenstrahlen, **36,** 572, 1927.

(12) SCHRÖDER, R. (Kiel), and JACOBI, H.: Ueber die Wirkung des Lipiodols auf die Genitalschleimhaut und das Beckenbauchfell. Arch. f. Gynäk., **142,** 514–529, 1930.

(13) SCHULTZE, G. K. F.: Gefahren und Schädigungen bei der Röntgenkontrastdarstellung der weiblichen Genitalien. Arch. f. Gynäk., **144,** 573–575, 1931.

(14) NEUSTAEDTER, THEODORE, EHRLICH, DAVID E., DU BOIS, JOHN COERT, and BLALOCK, GEORGE R.: A New Contrast Medium for Use in Uterosalpingography: Preliminary Report. RADIOLOGY, **21,** 568–572, December, 1933.

(15) TITUS, PAUL, TAFEL, R. E., MCCLELLAN, R. H., and MESSER, F. C.: A New, Non-irritating Opaque Medium for Uterosalpingography. Am. Jour. Obst. and Gynec., **36,** 889–898, November, 1938.

(16) MORSE, A. H., and RUBIN, I. C.: Study of the Comparative Radiopacity of Certain Contrast Media in the *Macacus rhesus*. To be published in Am. Jour. Roentgenol. and Rad. Ther.

(17) RUBIN, I. C.: Subphrenic Collection of Lipiodol Following Injection into Fallopian Tube, with Observations on Reverse Gravitation of Pelvic Exudates and the Genitophrenic Syndrome in Women. Am. Jour. Obst. and Gynec., **31,** 230–240, February, 1936.

HODGKIN'S DISEASE[1]

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ALTHOUGH it is true that in the past few decades, Hodgkin's disease (lymphogranulomatosis) has been extensively discussed from the etiological, diagnostic, and therapeutic viewpoints, there is still a definite indication for reviewing and discussing a group of cases that have been encountered in office, as distinguished from institutional, practice. They have certain individual and group characteristics worthy of note. The opportunity for leisurely and extensive study is not so readily available, as they often appear and disappear in a most irregular fashion; they may come to one's attention following therapeutic efforts by others; then too, the disease itself is not a very common one in private radiological practice.

The accompanying table (Table I) indicates that during the period from 1926 to 1936, eight cases of Hodgkin's disease were treated. The ratio of male to female patients treated in this series is at variance with the percentages met with in larger series of cases. Usually the proportion, however, is about two to one. The age distribution in this group was rather typical. The occurrence of the primary lesion was for the major portion characteristic, but there were certain individual features. In no case was the primary splenic form of Hodgkin's disease demonstrated. It has been repeatedly affirmed that the presence of inguinal adenopathy practically always suggests abdominal disease, while cervical or axillary involvement accompanies mediastinal disease. In Case 5, there was axillary adenopathy with no roentgenographic indication of mediastinal disease, while in Case 6, with cervical involvement, the mediastinal lymph nodes were slightly involved but only terminally.

All the cases that were studied terminated fatally, although Case 2 lived six years after the onset of therapy and nine years after the first sign and symptoms were noted. If the anamnesis of Case 8, M. A., can be accepted, the span of life in this patient from the beginning of the objective sign was about sixteen and one-half years. According to Flax's report (1), the average duration of life in a majority of cases since the beginning of the disease was twenty-seven and one-half months. Twenty-five per cent of the cases he reviewed reached an average duration of fifty-four and two-tenths months. Craver reports (2), however, 17 per cent of five-year or longer survivals, which appears to be within the realm of possibility. In the author's group, palliation prolonged the life of one case so that the survival of six years was equivalent to 12 per cent of the series.

Rosh (3), quoting Reisner and Brada, states that 74 per cent of cases present bone involvement with subjective signs. In the series reported, no symptoms referable to bone involvement were present; therefore, roentgen examination of the osseous system was dispensed with. However, the routine roentgen examination of the thorax did not reveal any involvement of the ribs, vertebræ, or humeri.

Beside the objective manifestations, cough of either productive or dry type was encountered when the mediastinal or hilar area was involved. Progressive anemia, loss of weight, and asthenia were other familiar subjective signs during the progress of the disease.

The most interesting and dramatic complication in the author's series occurred in Case 8, which is herein reported in detail.

Case 8. M. A., lawyer, aged 32, was apparently in good health in spite of a slight enlargement of the submental and left cervical lymph nodes which he had noticed for a period of 15 years. During the past five years these nodes appeared to increase and spontaneously decrease in size from time to

[1] This is one of a series of papers contributed by friends and former pupils of I. Seth Hirsch, M.D.

time. Within several weeks of his seeking medical advice, the submental lymph nodes showed a gradual increase in size; they were painless, slightly mobile, and comparatively hard to touch. There was no local heat or increase of the body temperature. Several nodes of the anterior chain of the left cervical region were readily palpable. Examination made by several eminent pathologists of an excised cervical lymph node revealed a morphology strongly characteristic of Hodgkin's disease. The submental and cervical areas were irradiated between Nov. 4, 1936, and Dec. 11, 1936, with complete regression of the enlarged cervical nodes and partial diminution in size of the submental nodes. A total of 800 r was administered. Recurrence of the lymph node enlargement in the areas irradiated took place within several weeks.

During February, 1937, the patient began to experience severe headaches and vertigo, whereupon physical examination revealed a choked disc on the right side. The symptoms were progressive and he was finally admitted to a hospital, in Baltimore, for careful work-up and diagnosis of the intracranial symptoms. A craniotomy, done in June, 1937, revealed a large granulomatous mass involving the meninges and right frontal lobe. Complete excision was done. Histological examination of the specimen revealed a "tuberculoma." The patient went through a stormy period with progressive disorientation, finally into coma, and died in August, 1937.

Comment.—The histological findings in a specimen removed from the brain stimulated a review of the slides made of the cervical node removed during February. Some of the pathologists were of the opinion that the lesion indicated Hodgkin's disease, while one suggested the possibility of lymphosarcoma. There is a mute question difficult to answer in this case: was this patient suffering from Hodgkin's disease with a complication of tuberculosis of the meninges and the brain, or was the initial lesion primarily tuberculosis? The response to irradiation, as well as the his-

TABLE I

	Sex	Age	Primary Site	Duration of Symptoms before Therapy	Biopsy and Laboratory Findings	Therapy	Course Following Therapy	Condition before Exodus
1. E. C.	M	10	Mediastinal Area	1 month	Positive. Anemia Eosinophilia 2 to 15 per cent	Irradiation from 11/1926 to 2/15/27	4 months; death	Abdominal node involvement and ascites
2. M. S.	M	38	Right Cervical Area	3 years	Positive. Anemia Eosinophilia one per cent	Irradiation from 11/1926 to 5/1932	6 years; death	Mediastinal and abdominal nodes involved
3. A. C.	M	32	Inguinal, Axillary, Cervical Areas	5 months		12/10/28	Did not continue	
4. J. K.	M	44	Cervical, Mediastinal, Hilar Areas	7 weeks		6/7/29	Did not continue	
5. J. B.	M	46	Right Axillary and Inguinal Areas	6 months	Positive	Irradiation from 8/1/29 to 8/19/29	No mediastinal involvement	
6. D. S.	F	52	Left Cervical Area	1 year	Positive. Anemia	Irradiation from 10/5/33 to 10/12/35. Transfusions	2 years; death	Mediastinal, inguinal, and abdominal nodes involved
7. J. D.	M	18	Left Supraclavicular Area	1 year	Positive. Anemia	Irradiation from 6/9/34 to 12/7/34. Two transfusions	3 months; death	Mesenteric, retroperitoneal node involvement
8. M. A.[1]	M	32	Submental Area	15 years	Positive. Anemia Terminally	Irradiation from 11/14/36 to 12/11/36	8 months; death	Meningeal granuloma

[1] See Text.

tological findings, would strongly suggest Hodgkin's disease.

Comment on Radiation Therapy.—The areas primarily involved or those that eventually recurred were treated with high voltage irradiation consisting of, in some cases, 180 kv., 4 ma., 0.5 mm. copper and 1.0 mm. aluminum filters, 50 cm. distance. In a few cases, 190 kv., 4 ma., 0.5 mm. copper and 1.0 mm. aluminum filters, 50 cm. distance were employed. Between 175 and 200 r were administered per treatment and per area. The results did not vary appreciably, no matter what kilovoltage was employed. As a rule, treatment was discontinued when the nodes in the area irradiated revealed complete regression, so that at no time was the full quota of skin tolerance approached.

Prophylactic irradiation, recommended by some therapists, was not employed. Ratkóczy (4) stresses the uselessness of irradiation during the periods of regression. Furthermore, in view of the presence of anemia in patients with lymphogranulomatosis intensive and persistent irradiation is obviously contra-indicated. Panteleroentgenotherapy as a means of combating Hodgkin's disease is rejected by Gilbert (5), who prefers local therapy.

Craver (2) discusses the question of choice between 140 kv. x-rays and 200 kv. x-rays as arbitrary with the therapist. He attests, however, that while regression of superficial nodes can be brought about by low kilovoltage, a 200 kv. technic is preferred for deep-seated lesions in Hodgkin's disease so as to spare the skin.

Desjardins (6) dislikes high kilovoltage (200 kv.) because, first, it may affect the patient adversely if repeated many times, and, second, it leaves no recourse to further therapy if lesions recur.

In the series presented herein, no contraindication to the use of high voltage therapy was observed in view of the paucity of the symptoms which necessitated only suberythema doses for the local recurrences or exacerbations.

It is to be regretted, however, that when the terminal stage of the disease was reached the nodes appeared to have become radioresistant. In some instances one is reminded of a forest fire where, as soon as one area is extinguished, another site of conflagration is discovered, each one in turn becoming more resistant to quenching.

SUMMARY AND CONCLUSIONS

Eight cases of Hodgkin's disease are reported, five of which were observed until their demise.

Two of the patients lived nine and sixteen years, respectively, from the beginning of the primary lesion.

One unusual case with a meningeal complication is reported in detail.

Irradiation served to delay the ultimate result. Involvement of the abdominal nodes was, as a rule, a terminal manifestation and hastened exodus.

77 Park Avenue

BIBLIOGRAPHY

(1) Flax, C. H.: The Treatment of Lymphogranulomatosis with Roentgen Rays. Strahlentherapie, **54,** 245–260, 1935.

(2) Craver, Lloyd F.: Local and General Irradiation in Hodgkin's Disease. Radiology, **31,** 42–47, July, 1938.

(3) Rosh, Rieva: Vertebral Involvement in Hodgkin's Disease; Report of Three Cases. Radiology, **26,** 454–458, April, 1936.

(4) Ratkóczy, N.: Destructive Doses in Roentgen Therapy of Lymphogranulomatosis. Strahlentherapie, **56,** 325–336, 1936.

(5) Gilbert, René: The Evolution of Radiotherapy, During the Past Ten Years, in the Treatment of Certain Generalized Affections. Radiology, **30,** 191–195, February, 1938.

(6) Desjardins, A. U.: Radiotherapy for Hodgkin's Disease and Lymphosarcoma. Jour. Am. Med. Assn., **99,** 1231–1236, Oct. 8, 1932.

THE INDIRECT RADIOGRAPH[1]

By A. BOUWERS, D.Sc., *Eindhoven, Holland*

From the X-ray Laboratories of the N. V. Philips' Gloeilampenfabrieken

DURING the last few years increasing interest has been shown in the indirect radiograph in which the film is not obtained by direct contact with the intensifying screen, but from an image on a fluorescent screen by means of a lens or system of lenses. Up to the present, the aim in the application of this indirect method has been merely to replace radioscopic examination by a method which was less dangerous, and, in addition, to offer certain other advantages, such as better documentation. In this connection we refer, for example, to the publications by Janker (1) and de Abreu (2).

Naturally, such means as were available have been employed in the practical application and the method has been adapted as well as possible to these means. At this stage it may perhaps be useful to try to find the conditions under which this method—which in the future will undoubtedly be found to be of great significance—gives the best result. The outcome of a number of tests and calculations for the purpose of finding these optimum conditions form the subject of this paper. Here we have thought not only of replacing the radioscopic image by the small film, but have also investigated the extent to which the reduced radiograph is inferior in quality to the radiograph by the usual method.

1. *The Optimum Radiograph: The Law of Uniformity.*—The indirect method is distinguished from the direct one through its exposure by means of a lens, which naturally entails a loss of light. Though it is true that because the reduction is the size of the image the amount of light per unit of surface area in the image is increased again, we shall see later that in the reproduction a considerable loss in light intensity occurs and that even with the fastest lenses the light intensity in the image is still only about one-tenth of that obtained with direct contact between screen and film. This loss of light intensity is mainly responsible for the loss in quality in the reduced radiograph. Now we know that for the normal radiograph of a moving object the recording conditions are best when the three causes of unsharpness—geometrical unsharpness (U_g), unsharpness due to movement (U_m), and unsharpness attributable to the screen (U_s)—are equal (3, 4):

$$U_m = U_s = U_g \qquad (1)$$

Here mean values are to be taken for the values U_g and U_m which are not constant for the whole image. We have called this condition the law of uniformity (3). Actually, it is based on the fact that the product of $U_m \times U_s \times U_g$ is a measure of the amount of light reaching the film and that, therefore, at a given value of the product, a given density is achieved. Now the sum of three figures with constant product is smallest when the figures are equal, as can easily be checked by figures taken as examples. This demonstrates the law of uniformity.

Strictly speaking, Condition (1) applies only when an x-ray tube with rotating anode is used (which is certainly preferable for this special purpose) and when the focal screen distance remains constant. By varying this distance, for instance to one-half, a four times greater intensity of radiation is obtained at a twice greater geometrical unsharpness, U_g. The product, $U_m \times U_s \times U_g$, therefore, becomes only twice greater at this reduced distance and the intensity quadruples. This is a reason for making the distance as small as possible when using a rotating anode: in fact, as small as other considerations, such

[1] This is one of a series of papers contributed by friends and former pupils of I. Seth Hirsch, M.D.

as distortion and enlargement of image, permit.

Further, in the deduction of the law of uniformity no mention has been made of tube voltage or contrast, but the conclusion is independent of all these circumstances; it applies generally under the said conditions.

in order to obtain a greater sensitivity, so that U_s is increased. Finally, the unsharpness in the small image is about 2.7 times greater than in the direct image.

The loss in sharpness of the image is determined by the ratio of the amount of light per square centimeter in the reduced image, I', to the amount of light, I, that

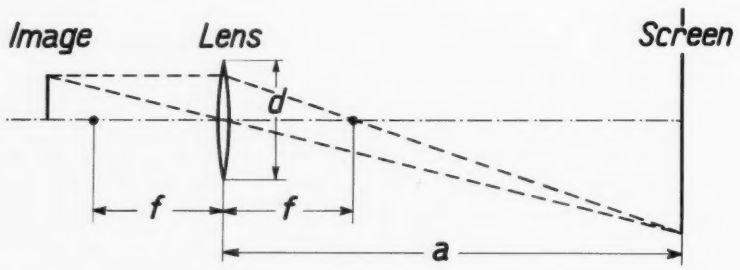

Fig. 1.

Also, in the case of indirect reproduction there is no change in the law of uniformity. We will take as a basis a radiograph made according to the ordinary method, which we will assume to be optimum, and substitute for the double intensifying screen a single screen which is recorded on film by means of a lens. If we keep to the same x-ray tube and also the same screen, we shall, for instance, have to expose twenty times longer to give the film sufficient density. As a consequence, the unsharpness due to movement will be greatly increased, to say nothing of the large dose of rays to which the patient is exposed, and the fact that the tube will probably not be able to withstand the much increased load. We must, therefore, select another procedure and the law of uniformity shows the way. We must arrange once again that approximately $U_m = U_s = U_g$, but in this case the product must be twenty times greater. We must multiply each of the factors U_m, U_s, and U_g by $\sqrt[3]{20}$ = about 2.7; and, therefore, we select a tube with a wider focus to increase U_g, increase the exposure duration 2.7 times to increase U_m, and use a screen with a coarser grain and a thicker layer than our intensifying screen

would be present per square centimeter of film in the case of a direct image. The factor k, which indicates the increase in the unsharpness of image, is determined by

$$k = \sqrt[3]{\frac{I}{I'}}. \qquad (2)$$

There are, it is true, some other factors which can influence the density of the reduced image, but these are much smaller than the above-mentioned ones and, moreover, partly compensate each other. The influence of these factors is all the more negligible since only the cube root counts. For instance, one of these factors is that the density in the reduced image, in connection with projection, must preferably be much less than that to which we are accustomed in the direct image. We also have assumed that the film sensitivity is the same in both instances: this need not be the case, for fundamentally the grain may be larger in the direct image than in the case of the reduced image, as we shall see later. On the other hand, however, the color of the light with the usual intensifying screens is not as suitable for modern films as that emitted by coarse-grained screens, to which we have to give

preference for indirect radiographs. Experience has taught us that in both cases practically the same sensitivity may be reckoned with when, for the indirect exposures, Kodak Super XX panchromatic, Agfa Isopan ISS 22/10° or Agfa Isochrom 17/10° DIN is used.

The difference in gradation also plays a part, as this influences the permissible voltage on the x-ray tube: a higher voltage is permissible with films having a steep gradation (5).

It is advantageous to select a somewhat higher voltage than would be favorable with direct exposures, as here increased sharpness is obtained with a certain loss of contrast: through the higher voltage, the x-ray output is increased and, consequently, the produce $U_m \times U_s \times U_g$ becomes smaller for a given density. This means, in spite of a much greater unsharpness of the indirect image as compared with the direct image, an increase in perceptibility. Due to such a compromise, the factor I/I' is decreased. We also gain light intensity in the reduced image by reduction of the focus–screen distance, as explained under Section 1. A compromise which gives about the optimum result, is obtained if with the indirect exposure the focus–screen distance is reduced to one-half; and if, at the same time, the voltage is increased about 30 per cent. The sharpness of the obtainable result is then about doubled.

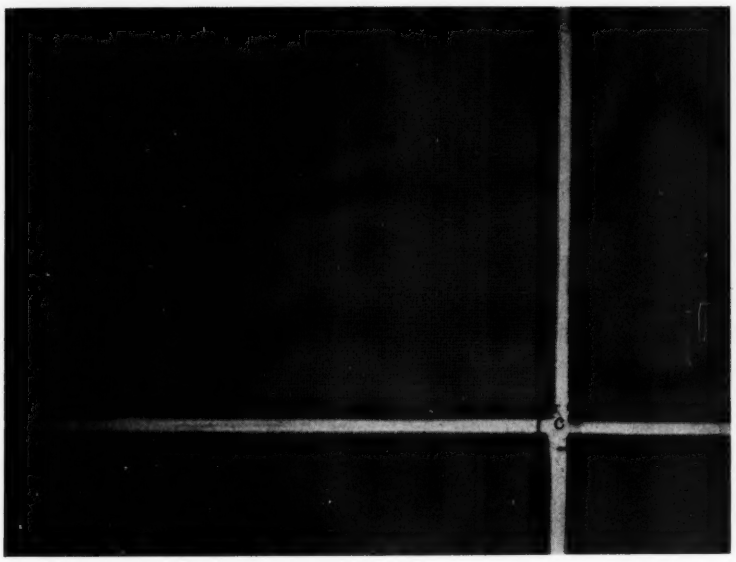

Fig. 2.

2. *Illumination Loss with Indirect Recording.*—We can easily calculate the ratio I'/I. Assume the diameter of the lens to be d, and the focal distance to be f (Fig. 1). The aperture, d, is in the case of the fastest commercial lenses $f/1.5$. Let the screen–lens distance be a. The relation of the light passing through the lens to the total amount of light emitted by the screen is

$$\frac{\pi d^2}{4} : 2\pi a^2 = \frac{d^2}{8a^2}.$$

If the reduction is n-times, then the surface area over which the light passing through the lens is distributed becomes $1/n^2$ times that of the screen and consequently the light intensity in the reduced image becomes $n^2d^2/8a^2$ times that in a

film immediately in contact with the screen and $n^2d^2/16a^2$ times that in a film between two screens. Now n is approximately

Fig. 3.

a/f, so that the last result may also be written in the form

$$\frac{I}{I'} = 16\left(\frac{f}{d}\right)^2 \qquad (3)$$

Applying (2), we find for the increase in unsharpness the factor:

$$k = \sqrt[3]{16\left(\frac{f}{d}\right)^2} \qquad (4)$$

If we halve the distance and increase the voltage 30 per cent with the indirect exposure, to approach optimum conditions as discussed above, this becomes:

$$k' = \sqrt[3]{4\left(\frac{f}{d}\right)^2} \qquad (4\text{-}a)$$

Now the value of f/d is generally not smaller than 1.5 in the case of camera lenses. Special lenses with larger aperture exist, however: the R-Biotar (6) of Zeiss has an aperture $f/0.85$, with f = 4.5 or 5.5 cm. Its unsharpness is, however, larger than with the commercial lenses mentioned below and certainly too large for images greater than 16×16 mm.^{2} near the focal plane. With a lens of this aperture ratio the reduced image would, according to Formula (4), have an unsharpness $\sqrt[3]{11.5} = 2.2$ times as great as would be possible with the ordinary method. Therefore, under the given circumstances a tube with a rotating anode would give about the same degree of sharpness in a reduced image as a tube with stationary anode by the ordinary method, since the comparative unsharpness for these two types of tube is about this amount. This rule also applies when using commercial lenses with an aperture of $f/1.5$ when the distance is halved at the same time and the voltage increased about 30 per cent, in which case Formula (4-a) may be applied.

3. *Unsharpness of Film and Lens.*—There is an additional unsharpness in reproduction, owing to the fact that the lens does not reproduce a point as a point but as a circle (circle of confusion) and that the film does not have an infinitely small grain. Strictly speaking, it would be necessary to examine in what way these last two magnitudes vary with the speed of the lens and with the sensitivity of the film in order to arrive at a more complicated law of uniformity for the reduced image. However, these relations are not so simple. For the present the best thing is to require that the unsharpness produced by imperfect lens correction and grain of the film shall be small compared with the unsharpness that would be produced by ideal reproduction due to the causes already mentioned.

As regards grain, we have benefited by the rapid development of miniature film during the last few years due to the demands of miniature photography. Many firms now carry fine-grain stock. With suitable methods of development, it is easy enough to obtain a fineness of grain that will satisfy the above requirements. On measuring the size of grain of various

films, we found values varying between a few micra and about 25μ. When suitable material is used, the size of grain need not exceed 10μ.

If we take the unsharpness of the normal x-ray image as 1 mm. (this is already low when the indirect method is concerned), after being reduced fifteen times the unsharpness in the reduced image is approximately 0.07 mm. = 70μ. The requirement that the size of grain shall be small as compared with the other unsharpnesses is thus met.

In regard to faults due to imperfect lens correction, we must be more careful. The data measured in the case of two very good lenses are shown in Table I. The circle of confusion was measured by the Foucault method and an effective value established as well as possible. This effective value is then about the same as that calculated from the resolving power, which is determined by the maximum density of lines visible in the photograph of several wire meshes. This density of just visible lines is also indicated in Table I. The Xenon lens shows rather strongly the phenomenon known as coma, as a consequence of which the number of visible lines in the corner of the field is not greater than with the Sonnar, although the effective circle of confusion is much smaller.

It follows from the Table, that the lens' faults alone produce an unsharpness of the same order as that which would occur with ideal reproduction. Lenses with considerably greater errors in correction would be useless. When examining a lens for this special purpose the tests should preferably be made in light of the color of the screen itself. The best thing to do is to work directly with the fluorescent light, for slight deviations in color may have a considerable effect on the lens correction, as lenses are usually corrected for two very definite colors and often show considerable deviations for other colors. Thus, for instance, the circle of confusion in the case of the above-mentioned Sonnar lens was about twice as great in blue light as in white light.

Figure 2 gives an example of an exposure of a number of wire meshes to establish the resolving power or the degree of unsharpness, of the lens–screen combination. Here the wire meshes are laid on the fluorescent screen so that the latter serves as an illu-

TABLE I

	Relative Aperture	Mean Effective Circle of Confusion		Visible Number of Lines per mm. in Reproduction of Wire Mesh	
		Approximate Axis	In Corner of 24 × 32 mm. Field	Approximate Axis	In Corner of 24 × 32 mm. Field
Sonnar	1:1.5	30μ	50μ	30μ	20μ
Xenon	1:1.5	20μ	30μ	40μ	20μ

minant. If the meshes are placed behind the screen so that the x-ray image of the meshes are photographed, the same method may serve for establishing the unsharpness of the screen.

4. *Most Favorable Conditions for Indirect Exposures.*—After what has been stated heretofore, we can now draw some further conclusions regarding the most favorable conditions for making indirect exposures.

In the first place, we find that the aperture of the lens when maintaining the conditions of sharpness, must be as large as possible, as this magnitude, according to Formula 3, determines the brightness of the reduced image and, according to Formula 4, the sharpness of it.

As the unsharpness of the lens is of the order of 30μ and the grain of the film can easily be maintained under this value, we may require that for the degree of photographic reduction used the reduced unsharpness on the film shall amount to a few times 30μ. If this were not so, the unsharpness of the lens would have an unfavorable influence. Now by utilizing the standard miniature size of the Leica, Kodak Retina, Contax, etc., the size of the film is about 24 × 32 mm.[2] This signifies that an image 30 × 40 cm. would be reduced 12.5 times in order to become this

size. An unsharpness of about 1 mm. on the screen must be reckoned with, so that the reduced unsharpness would amount to 80μ. The figures show that the size of 24 × 32 mm.^{2} is large enough to produce all the details present in the radiograph, provided the lens unsharpness does not exceed 30μ. In regard to grain, even the sub-standard film is preferred by Russell Reynolds (7) in his cinema technic. It must be observed, however, that at least in regard to stationary images, the grain is already troublesome, causing an annoying effect during observation. This effect is considerably less in cinematography. However, in the case of these very small radiographs, the faults of the lens begin to become serious unless special lenses are used, having a circle of confusion in the whole image not exceeding about 10μ.

Finally, we can determine from the above considerations approximately the best exposure conditions for a lens of an aperture $f/1.5$. Taking, as example, a lung at a distance of 90 cm. lens–screen. Abreu (2) and Holfelder (8) recommend focus–screen distances of, respectively, 60 and 70 cm. and arrive at the following exposure conditions: size, 24 × 32 mm.^{2}; tube voltage, 80 kv. peak; focus of the Rotalix tube, 2 mm.; Agfa Isochrom film; a zinc sulphide screen with an unsharpness of approximately 0.6 mm.; time, 0.1 sec.

Figure 3 shows a lung radiograph taken under the above conditions with an $f/1.5$ lens.

SUMMARY

The indirect radiograph, obtained by means of a reduced picture of the screen image with a lens, is compared with the direct exposure. The result is that, with approximately equal contrasts, the unsharpness of the indirect method is approximately $\sqrt[3]{I/I'}$ times greater than in the case of the direct exposure, when I/I' is the ratio of the light intensity, I, in the large image to I', that in the reduced image. This ratio can easily be calculated from the aperture d/f of the lens, *viz.*:

$$\frac{I}{I'} = 16\left(\frac{f}{d}\right)^2.$$

The lens must, therefore, have as large a relative aperture as possible without the reproduction faults exceeding the indicated limits.

The photographic quality of the indirect image is approximately equal to that of the direct image (ordinary technic) when for the indirect image a tube with a rotating anode is used and for the direct image a tube with a stationary anode.

The most suitable size is approximately that of the miniature camera, 24 × 32 mm.^{2}, when a lens is used with an effective circle of confusion not larger than about 30μ.

The conditions are indicated under which with a lens having a relative aperture of $f/1.5$ and the application of the rotating anode, approximately the most favorable exposure is obtained.

REFERENCES

(1) JANKER, R.: Deutsche med. Wchnschr., **58,** 1094–1096, July 8, 1932.
(2) DE ABREU, M.: Ztschr. f. Tuberk., **80,** 70–91, 1938.
(3) BOUWERS, A., and OOSTERKAMP, W. J.: Fortschr. a. d. Geb. d. Röntgenstrahlen, **54,** 87–91, July, 1936.
(4) BOUWERS, A.: Fortschr. a. d. Geb. d. Röntgenstrahlen, **56,** 148, 149, July, 1937.
(5) ALFTER, J., and OOSTERKAMP, W. J.: Fortschr. a. d. Geb. d. Röntgenstrahlen, **55,** 609–612, June, 1937.
(6) ZEISS, CARL (London): Jour. Sci. Instr., **11,** 365, 1934.
(7) REYNOLDS, RUSSELL J.: Jour. Inst. Elec. Eng., **79,** 389, 1936.
(8) HOLFELDER, H.: Fortschr. a. d. Geb. d. Röntgenstrahlen, **58,** 181–187, August, 1938.

COLLECTIVE FLUOROGRAPHY[1]

By DR. MANOEL DE ABREU, *Rio de Janeiro, Brazil*

From the Medical Academy

TWO methods of x-ray analysis have been available for the thoracic survey of large groups of individuals. One, radiography, is expensive and impractical, while the other, fluoroscopy, requires the services of a considerable number of skilled specialists (about 150 per million examinations per annum).

Fig. 1. The first collective apparatus, installed by Dr. Manoel de Abreu in May, 1936, at the Hospital Allemão, Rio de Janeiro.

The method of fluorography or fluoroscopic screen photography, or indirect radiography, which has also been called roentgen photography, does, however, permit practical chest surveys of large groups effectively and at a low cost.

Indirect radiocinematography, an expensive method, is still in the experimental stage and may eventually be useful for certain types of research and teaching, but it is not to be confused with the method of roentgen photography utilized by the writer for tuberculosis prophylactic surveys.

Though fluorography was attempted long ago by many workers, the early results were unsatisfactory. Blyer (1896), MacIntyre, Porcher, Köhler, Lomon and Comandon, Cole, and others experimented with the method. In 1918, and later, in 1924, we attacked this problem diligently but were unable to obtain decisive or practical results. Then most of the work was directed exclusively to the solution of the problem of radiocinematography. More recently this has been accomplished by Reynolds, Djean, and Janker.

Fig. 2. One of the first films obtained by collective fluorography in May, 1936. Large infiltration on the right side and slight infiltration on the left.

We inaugurated the first installation of fluorographic apparatus for the purpose of carrying out a collective thoracic survey at the German Hospital of Rio de Janeiro, in 1936; thus our priority cannot be contested.

In 1937 three additional centers were operating, one in the Public Health Department of Rio de Janeiro, one in the Navy Hospital, and one in the Public Health Department of Victoria.

At present,[2] due to the great interest

[1] This is one of a series of papers contributed by friends and former pupils of I. Seth Hirsch, M.D.

[2] Early in 1939.

aroused by this work, we have 25 installations in Brazil and many others in Argentina, Chile, Uruguay, Germany, France, etc. The method has also been carried to the United States by Dr. D. O. N. Lindberg, of Decatur, Illinois, who, after his visit to us late in 1937, introduced fluorography in his sanitarium. Our attempts, in 1937, to interest American manufacturers in the construction of this apparatus were unsuccessful.

To-day, three years after the first practical results were achieved, collective fluorography is universally accepted. At the beginning of this work, there were many roentgenologists who expressed themselves in opposition to the procedure but have since become convinced as to its practicability.

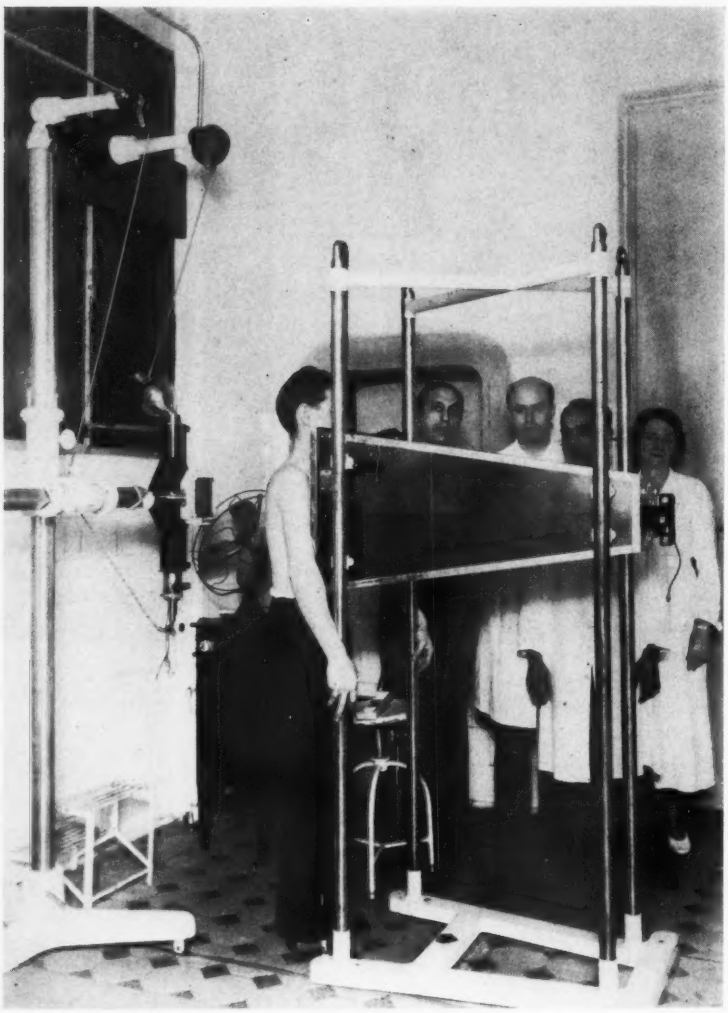

Fig. 3. Roentgenphotographic apparatus "Manoel de Abreu" at Rio de Janeiro Public Health Department (June, 1937).

The objections with which we had to contend at the beginning were principally that since the initial roentgenological indications of tuberculosis are frequently very vague, fluorography would not show sufficient detail to demonstrate early tuberculosis, and that fluoroscopy in various planes was essential to diagnosis. Also, the wear and tear on x-ray tubes from the exposures on a large scale would make the procedure expensive.

In answer I quote from Professor Hans Holfelder (Introductory remarks of Professor Holfelder (17); "The present work of Dr. Abreu deserves the most widespread attention in the consideration of the question of a large-scale campaign against tuberculosis, since, for the first time in practice, a means has been found which, in my opinion, is capable of settling the problem of the proper position of roentgen diagnosis in the battle against tuberculosis." (*Fortschr. a. d. Geb. d. Röntgenstahlen*, **58,** 183, 1938.)

From our eminent colleague, Dr. R. Vaccarezza, President of the Argentine League Against Tuberculosis, we have received a communication from which we quote the following: "My gratitude was greater for having had the opportunity of learning your roentgen photographic method which I studied closely. Its application in social work I consider one of the greatest medical conquests of recent time." (Aug. 2, 1927.)

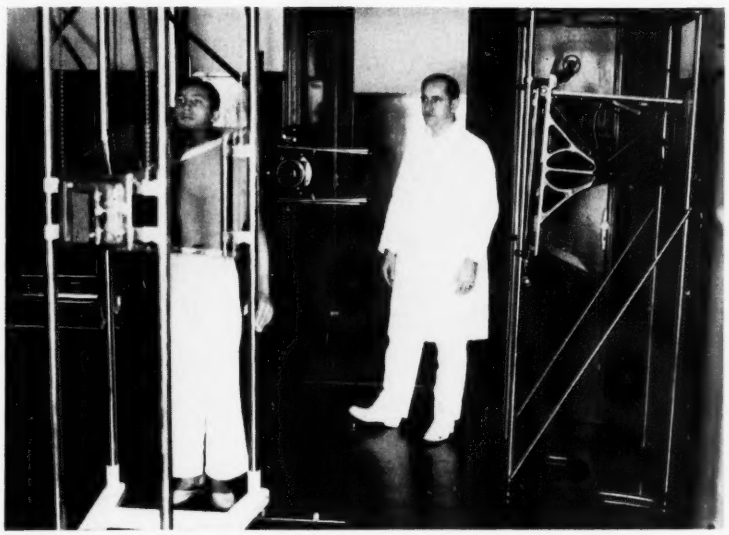

Fig. 4. The roentgenphotographic apparatus "Manoel de Abreu" at the Marine Center of Rio de Janeiro (August, 1937).

The technic used in the beginning of our work was as follows:

1. Four-valve rectifier apparatus.
2. Water-cooled 10 kw. line focus tube.
3. G. E. Patterson screen or Siemen's Super-astral screen without yellow varnish.
4. Objective Zeiss F 1.5.
5. Objective screen distance 90 cm.
6. X-ray tube—screen distance 60 cm.
7. Film, 35 mm. Agfa Isochrome F (28 Sch.).
8. Adult average chest, 50 ma., 80 kv., from 0.3 to 0.4 second.
9. Children, 100 ma., 100 kv., from 0.03 to 0.05 second.

Under these conditions, 35,000 fluoro-

graphs were made at the Public Health Department without damage to the tube.

Later modifications introduced are:

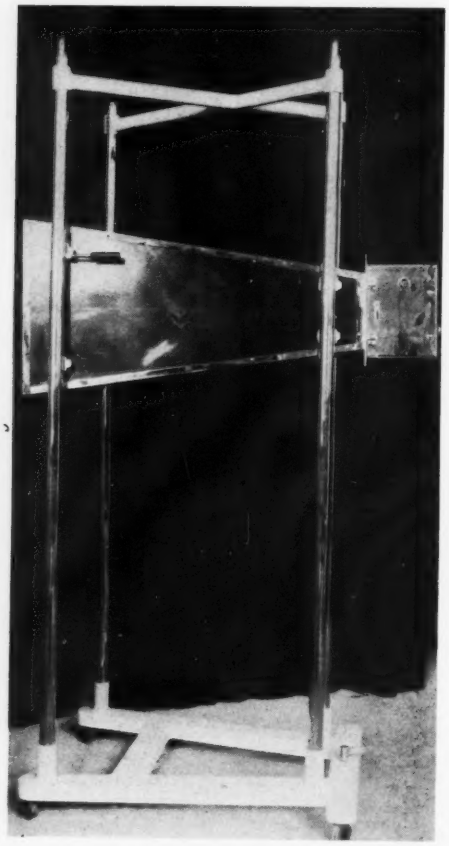

Fig. 5. The four-column apparatus built at the beginning of 1937, and inaugurated at the Health Department of Rio de Janeiro in June, 1937. Observe the special "collective" camera.

1. Kodak Super XX film (32 Sch.) or Agfa Isopan I.S.S. (31 Sch.).

2. Average adult chest, 50 ma., 55 kv., from 0.2 to 0.3 second.

3. Children, 100 ma., 50 kv., from 0.03 to 0.05 second.

With half wave apparatus, without rectification, we advise 70 kv., leaving the other factors unchanged.

The 35 mm. film size is preferred because it is easily available and inexpensive (about one cent per record). We are convinced that the small image, 2.4 cm. square, has a perfection of detail completely adequate for a diagnostic survey. Teleradiography and planigraphy must often be used as supplements for an individual case.

TABLE I.—COMPARISON OF EXPENSE OF THREE METHODS, FOR ONE MILLION EXAMINATIONS ANNUALLY

Radiography	$900,000
Radioscopy	340,000
Roentgen Photography	112,000

It is erroneous to believe that the greater detail obtainable with instantaneous teleradiography permits a finer diagnosis of tuberculosis. Our investigation of the limit of visibility of pulmonary lesions indicates that such visibility is mainly determined by radiogeometry, that is, by the angle of incidence of the radiation on the surfaces bounding different densities. If the incidence on the surface of a lesion is parallel or tangent, the visibility is clear; if, however, it is oblique or transverse with an obliquity greater than 22.5 degrees, the images are faint or may even vanish. Thus, in the best teleradiography, processes having a volume from 20 to 100 c.c. are not visible. We believe that frequently roentgen photography at various angles, such as anterior and posterior descending directions, has the advantage over teleradiography of showing faintly visible or invisible lesions.

The diagnosis of all or almost all cases of pulmonary tuberculosis by means of roentgen photography will assist in directing the social campaign against tuberculosis to meet the threat presented by the individual. The hazard, according to our conception, is dependent upon the relation between afflicted persons and children or adolescents.

The categories of decreasing hazards are three: (*a*) permanent contact (common domicile); (*b*) indirect or transitory contact; (*c*) absence of contact.

The approximate data for the city of Rio de Janiero are shown in Table II.

Thus, among the 687,000 persons living together with children, 2 per cent suffer from disease (13,740). Of these, 5,000 are the greatest hazard due to their close intercourse with children. The scale of hazard is as follows: mothers, 4.4.; female servants, 4.25; female relatives, 2.8; fathers, 2.8; male servants, 2.66; male relatives, 2.

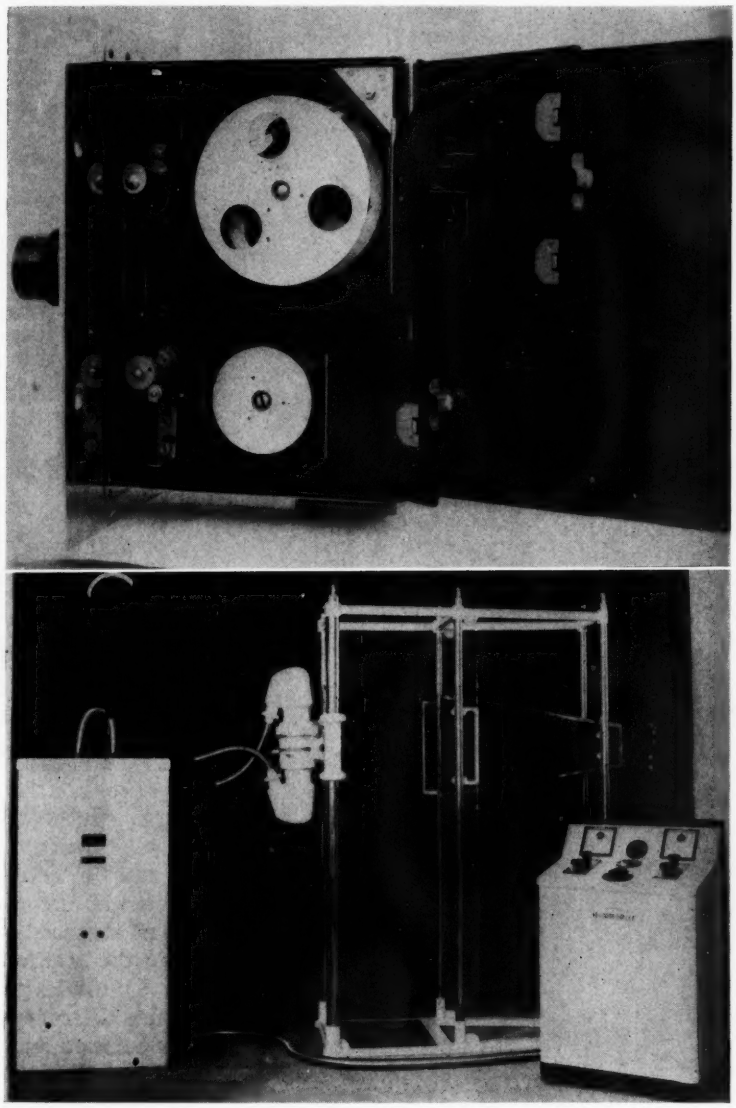

Fig. 6. The special "collective" camera used since 1937, with the 60 mm. film bobbin.
Fig. 7. The latest type of collective fluorographic apparatus with six columns, built in 1938, first installed at the Assistencia Municipal, Rio de Janeiro.

parents, 3,750 servants, and 5,000 relatives and friends. The females among these—mothers, servants, and relatives—present

The legislative measures which would

assure this campaign for a general survey should be based on the health examination already obligatory for those employed in certain occupations (collective). It would be sufficient if such health examinations were: first, required periodically with roentgen photography; second, extended to all workshops, homes, teaching, and sport centers; third, required not only of workmen, soldiers, teachers, students, and sportsmen, but also of members of their respective families and people sharing their homes.

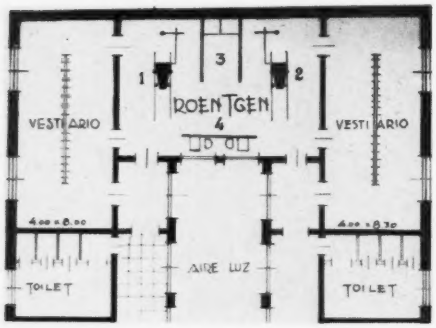

Fig. 8. The Argentine Center of Thoracic Census with two roentgenphotographic apparatus "Manoel de Abreu," under Dr. Vaccarezza's direction, to be inaugurated in Buenos Aires, May, 1939. 1 and 2, apparatus; 3 and 4, x-ray protectors.

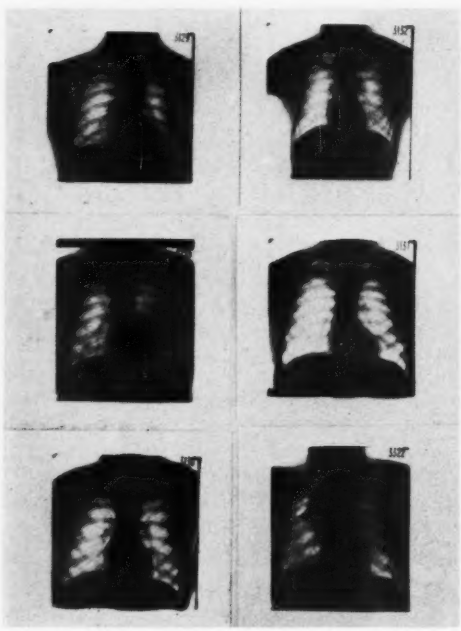

Fig. 9. Fluorographic prints, natural size.

TABLE II

Population	2,000,000
Minors	500,000 (25%)
Homes with children	125,000 (4 children each)
Parents	250,000 (2 to each home)
Servants in general	187,000 (1.5 per home)
Other adults in homes	250,000 (2 per home)
Total number of adults in homes	687,000 (5.5 per home)
Tuberculosis mortality	7,000 per annum
Number of tuberculous persons	40,000 (5.7 × mortality)

In this way there would be established a new and efficacious campaign against tuberculosis—a center for complete thoracic survey. Such a center should be equipped with social and statistical service and three roentgen photographic installations capable of completing 3,000 examinations daily, or 85,000 per month, or about 1,000,000 a year.

The thoracic survey center should, in addition to making the diagnosis, have control of all persons either tuberculous or suspected of being infected with the disease. Unknown tuberculous persons would then disappear. The social status of those individuals who present a hazard and of those who are menaced by exposure would be known to the health authorities. The thoracic survey would permit early diagnosis by means of roentgen photography of all the population and enable the dispensaries to operate with the utmost efficiency.

Systematic prophylaxis would at once solve the problem regarding suspects in our homes (servants and friends) who number 4,375. These persons would, without any expense, be summarily withdrawn from homes with children and adolescents. There would remain 2,500 tuberculous

parents with 5,000 children threatened or already infected. In this group, the children as well as their parents must be cared for with our present and future facilities.

The sanitation and prophylactic system based on hospital dispensaries failed, because a complete thoracic survey has until now not been made. Tuberculosis pro-

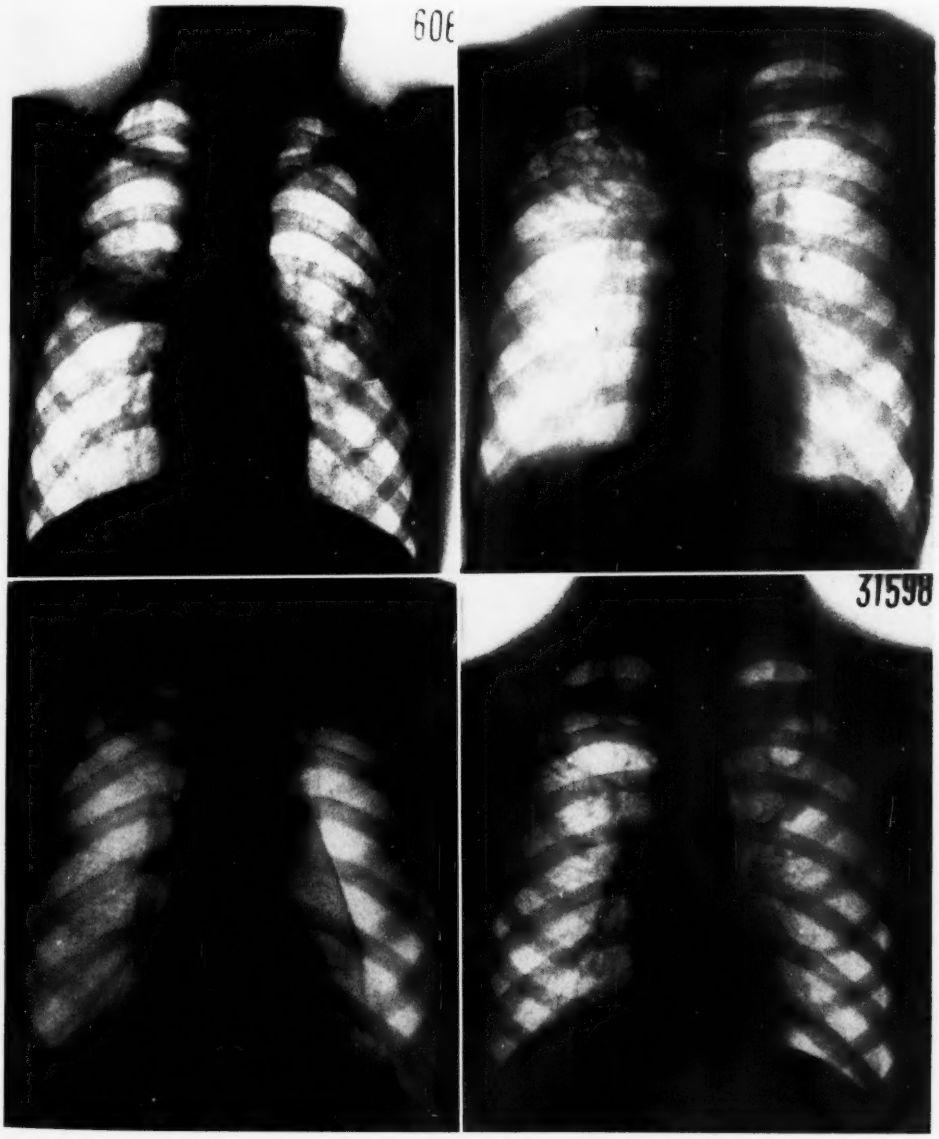

Fig. 10 (*upper left*). Enlarged fluorography. Infiltration in the region of the small right interlobar fissure. The left lung shows a small cavity between hilus and costal borders.

Fig. 11 (*upper right*). Fibroid tuberculosis of the upper right lobe. Undiagnosed, but discovered by the chest collective examination.

Fig. 12 (*lower left*). Left pneumothorax. The collapse therapy control at the Health Department of Rio de Janeiro is done only by fluorography, in two views: postero-anterior and right or left oblique.

Fig. 13 (*lower right*). A cavity in the upper right lobe. Another case of undiagnosed tuberculosis discovered by the collective examination.

phylaxis cannot be secured by treatment and isolation of a small percentage of tuberculous persons. On the contrary, it would be based on the discovery of all infectious foci followed by the isolation of all dangerous persons and protection of all those threatened and in a receptive condition.

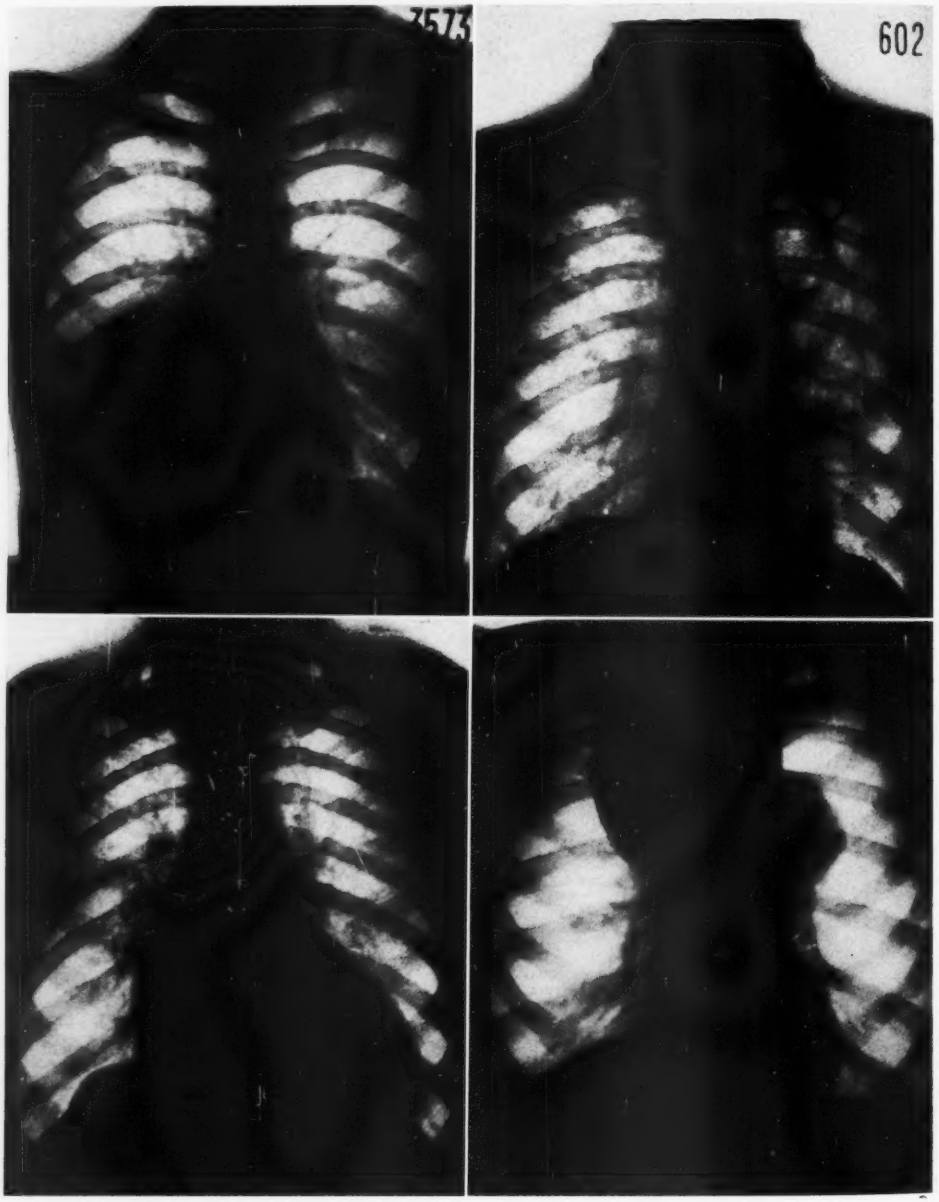

Fig. 14 (*upper left*). Consolidation at the right base.
Fig. 15 (*upper right*). Multiple cavities in left lung. Discrete foci on right side.
Fig. 16 (*lower left*). Aortic insufficiency; hypertrophy of the left ventricle.
Fig. 17 (*lower right*). Aortitis and large aneurysm of the ascending aorta revealed by collective fluorography.

We add that, in addition to tuberculosis, we simultaneously check the cardiovascular system, having in view the frequency of syphilitic aortitis in Brazil.

Systematic fluoroscopy as a control in collapse therapy has been abandoned by us and replaced with advantage by fluorography prior to insufflation. The description of the new method may be found in our book published early in 1938 (14).

Finally, we employ fluorography with planigraphy; in this way we can take a great number of sections at various angles with no expense.

REFERENCES[3]

(1) DE ABREU, MANOEL: Previous Note about Collective Fluorography. Med. and Surg. Soc. of Rio de Janeiro, July, 1936.

(2) Idem: Roentgenphotography: Process and Apparatus. Lung Tuberculosis: Social Census, Radiography, and Radioscopy. Collective Roentgenphotography. Rev. Assn. Paulista de med., **9,** 313–324, December, 1936.

(3) Report of Brasilian Tuberculosis Society on Manoel de Abreu's Method on Collective Thoracic Examination. September, 1936.

(4) DE ABREU, MANOEL: Roentgenphotography: Technic and Apparatus for Roentgenphotography and Thoracic Census. Día méd., **37,** Buenos Aires, 1937.

(5) MAZZEI, E. S.: The Roentgenphotography of Manoel de Abreu. Día méd., **37,** Buenos Aires, 1937.

(6) DE ABREU, MANOEL: New Ways on Tuberculosis Prophylaxis. Surg. and Phar. Jour., Rio de Janeiro, May, 1937.

(7) Idem: New Directions on Tuberculosis Prophylaxis. Día méd., **43,** Buenos Aires, 1937.

(8) Idem: Roentgenphotography: Technic and Apparatus. Día méd., **10,** 157–160, Feb. 28, 1938.

(9) MAZZEI, E. S.: Roentgenphotography. Argentina Med. Assn. Jour., Buenos Aires, May 20, 1938.

(10) VACCAREZZA, R.: Report for the Argentine League against Tuberculosis, Buenos Aires, 1937.

(11) DE PAULA, ALOYSIO, and BENEDETTI, F.: Present Orientation on Tuberculosis Dispensary. Hygienic Arch., **7,** November, 1937.

(12) FONTENELLE, J. P.: Annual Report of Federal District Public Health Department. Rio de Janeiro, 1937.

(13) BARRETO, J. DE BARROS: National Public Health Department Report, Rio de Janeiro, 1937.

(14) DE ABREU, MANOEL: Roentgenphotographic Thoracic Census (226 pages, 18 figures, 14 plates.) Published by O Globo Library, Porto Alegre, 1938 (delivered to the editor in July, 1937).

(15) Idem: Collective Fluorography. Seventh Pan-American Congress of the Havana Medical Association, January, 1938. (Introduced by Dr. José Pratts.)

(16) DE PAULA, ALOYSIO: Collective Roentgenologic Examination against Tuberculosis: Manoel de Abreu's Method and its Results. Lectures during the South American Medical Congress at Montevideo, January, 1938. Rev. de tuberc. d. Uruguay, **7,** 129–152, 1938.

(17) HOLFELDER, HANS: Presentation of Dr. Manoel de Abreu's Works on Collective Fluorography. Frankfurt, Germany, February, 1938.

(18) DE ABREU, MANOEL: Actual Situation of Roentgenphotography on Tuberculosis Prophylaxis. Official report presented to the First Regional Tuberculosis Congress, Rio de Janeiro, May, 1938.

(19) NEVES, JAYME SANTOS: Fight against Tuberculosis at Esperito Santos State and Manoel de Abreu's Method. First Regional Congress of Tuberculosis, Rio de Janeiro, May, 1938.

(20) DE ABREU, MANOEL: Technic and Apparatus for Collective Fluorography (Indirect Roentgenography). Ztschr. f. Tuberk., **80,** 70–91, 1938.

(21) Idem: Collective Radiophotography. Jour. de radiol. et d'électrol., **22,** 232–235, May, 1938.

(22) COELHO, JOAO: The Radiophotography of Manoel de Abreu. Presse méd., **46,** 1032, June 25, 1938.

MORE RECENT REFERENCES

(1) DE ABREU, MANOEL, DE PAULA, ALOYSIO, and BENEDETTI, F.: Argentine Archives of Respiratory Apparatus, special number on M. de Abreu's Roentgenphotography, May, July, 1938.

(2) DE PAULA, ALOYSIO: First Results of Roentgenphotography of Manoel de Abreu. Lecture at the Paulista Medical Society, Aug. 18, 1938.

(3) DE ABREU, MANOEL: Zwei Jahre kollektive Röntgenphotographie. Fortschr. a. d. Geb. d. Röntgenstrahlen, **58,** 574–587, December, 1938.

(4) DE PAULA, ALOYSIO, and BENEDETTI, F.: Epidemiological Dispensary. Roentgenphotography as Fundamental Work at the Tuberculosis Dispensary. Folha Med., Sept. 5, 1938.

(5) DE PAULA, ALOYSIO: Tuberculosis, 1 vol., 58 pages. S. P. E. S. Edition, 1938.

(6) PAULINO, FERNANDO: The Results of 15,000 Roentgenphotographs. Sixth National Congress of Medicine, at Cordoba (R. Argentine), Oct. 16–21, 1938.

(7) RONNEAUX, G.: Note on the Roentgenphotography of Manoel de Abreu. Bull. et mém. Soc. d'électro-radiol. méd. de France, **26,** 624–627, November, 1938.

(8) RONNEAUX, G., DEGAND, F., WATTEZ, E., and SAGET: Essay on the Application of Roentgenphotography in Pulmonary Stratigraphy. Bull. et mém. Soc. d'électro-radiol. méd. de France, **26,** 687–693, December, 1938.

(9) YANKER, ROBERT: Leuchtschirmphotographie Röntgenreihenuntersuchung. Leipzig, 1938.

(10) SAYE, L., and CAUBERRERE, N. L.: The Practice of the Method of de Abreu for the Collective Diagnosis of Tuberculosis. Presse méd., March 25, 1939.

(11) BARRETO, J. DE BARROS: Federal Health Services in 1938, pp. 52, 53, Rio de Janeiro, 1939.

(12) MARINHO, ABELARDO, DE PAULA, ALOYSIO, ASSIS, ARLINDO, CARNEIRO, FERNANDO, and PITANGA, GENESIO: Tuberculosis and Social Provision, p. 15, May, 1939.

[3] Collective Fluorographic Bibliography from 1936, 1937, to the beginning of 1938.

THE SPHINCTERS OF THE COLON[1]

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Translation by Theodore R. Frattalone, M.D., New York City

THE study of the sphincters of the colon, concerning which radiologists have contributed considerable data, is important from an anatomical, physiological, and pathological aspect.

From an anatomic and histologic standpoint, according to findings which I have previously presented, the sphincters of the colon are as shown in Figure 1.

(*A*) *Sphincter of Varolio* (Fig. 1-*a*).—Rutherford (1904), using anatomic specimens of animals which had succumbed to a violent death and of human cadavers (a few hours after death), was able to demonstrate that the ileocecal valve, in the fresh state had a very different aspect from that which the anatomist had described, that is, the ileocecal valve presented itself as a spherical eminence lacking a valve and frenula. The anatomic structure of this eminence, according to Rutherford, consisted of two layers of muscular fibers, circular and longitudinal, originating from the small intestine, an external third layer of circular fibers which formed a true sphincter, and, lastly, an internal fourth layer also made up of circular fibers (circular muscle layer of the mammillary eminence). At the apex of the valve these three circular layers unite in a single stratum of circular fibers.

I have been able to confirm Rutherford's findings and also to show that there are fibers which originate from the innermost layer of longitudinal fibers as above described, which, running an oblique course, meet the circular strata of the mammillary eminence where these terminate. Beside these of small intestinal origin, there are other fibers, also running obliquely, originating from the innermost part of the longitudinal fibers of the large intestine, which merge with the circular layer of the mammillary eminence. We shall refer to this when we consider the physiology of the sphincters.

(*B*) *Sphincter of Busi* (Fig. 1-*b*).—The sphincter of Busi is placed between the cecum and ascending colon, underneath the sphincter of Varolio. There is, in this area, an increase in longitudinal and circular fibers with a passage of fibers from the longitudinal layer to the circular layer. The vessels in this sphincter, as in the other sphincters, pass from the longitudinal to the circular layer to reach the submucosa and the mucosa.

(*C*) *Sphincter of Hirsch* (Fig. 1-*c*).—This sphincter, which is generally found in the proximal segment of the ascending colon, presents anatomically a complex of longitudinal and circular fibers of much heavier caliber than is present in the sphincter of Busi. The circular layer is reinforced by fibers placed in a ribbon-like fashion.

(*D*) *Sphincter of Cannon-Boehm* (Fig. 1-*d*).—This sphincter is found in the transverse colon at the junction of the proximal and middle thirds. However, it may vary in position in the same individual, and, therefore, one may assume the sphincter to have a relationship to the superior and inferior mesenteric plexuses. It presents no special muscular pattern.

(*E*) *Sphincter of Payr and Strauss* (Fig. 1-*e*).—This is found at the splenic flexure of the colon and does not present any histological difference in muscular structure from that of the colon proper.

(*F*) *Sphincter of Balli* (Fig. 1-*f*).—This sphincter, discovered by Balli, is found at the point where the descending colon becomes the sigmoid. Histologically,

[1] This is one of a series of papers contributed by friends and former pupils of I. Seth Hirsch, M.D.

it presents a different muscular pattern from that of the colon proper.

(*G*) *Sphincter of Moutier* (Fig. 1-*g*).—The sphincter of Moutier is found in the terminal colon between the pelvic colon and the rectum. This corresponds to the area (described by Beirne) at the level of the third sacral vertebra. Histologically, it is composed of interlaced longitudinal and circular fibers.

(*H*) *Sphincter of Rossi* (Fig. 1-*h*).—This sphincter is situated in the mid-portion of the sigmoid and does not present any particular muscular pattern.

From an anatomical standpoint, my work leads me to agree with the others that only four should have the right to be designated as sphincters because of their muscular pattern, namely, the sphincters of Varolio, Busi, Hirsch, and Moutier. Aloi, however, has found no modification from the normal structure of the colon in the regions of the sphincter of Busi and the sphincter of Rossi. There are thus only three organic sphincters: those of Varolio, Hirsch, and Rossi. The others, the sphincters of Cannon (even Arendt does not admit the existence of a true anatomical Cannon-Boehm sphincter, but indicates it simply as a point of contracture), Payr-Strauss, Balli, and Rossi, because of their muscular structure resemble the general muscular pattern of the colon, are to be called functional sphincters, that is to say, areas subject to contracture, but without any anatomic markings.

Aloi, continuing Balli's study, has verified the disposition of the fibers forming muscular plexuses which are evident in the anatomic structure of the ascending colon and terminal portion of the sigmoid, corresponding to the sphincters of Hirsch and Moutier.[2]

Thus, one is brought to define these constrictions, as the writer has already done, as sphincteric areas of the colon.

[2] There thus remains a difference of opinion between Aloi and the writer only concerning the sphincter of Busi, a discrepancy which, in all probability, is due to the peculiar conditions of the anatomical specimens studied.

According to Aloi, there is no difference in the number and size of the arterial vessels in the regions of the sphincters and the other parts of the large intestine. The author, on the contrary, has found an

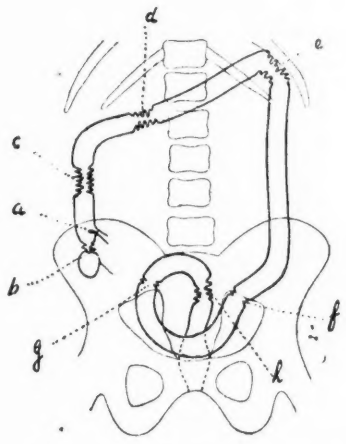

Fig. 1. Showing the sphincters of the colon, starting from the cecum: (*a*) Sphincter of Varolio (improperly called *ileocecal valve*); (*b*) sphincter of Busi; (*c*) sphincter of Hirsch; (*d*) sphincter of Cannon-Boehm; (*e*) sphincter of Payr-Strauss; (*f*) sphincter of Balli; (*g*) sphincter of Moutier; (*h*) sphincter of Rossi.

extraordinary richness of arterial capillaries extending to Auerbach's plexus in the muscular coat and to the plexuses of Meissner in the submucous area. Such an abundance of arterial terminations leads the author to classify these contracture zones of the large intestine as "the true, nerve hilus" of the large bowel, which when responding to stimuli, function actively in the intestinal movement.

From the physiologic viewpoint, credit is due to the radiologists for having studied the movements of the colon. Because of the rich innervation of the sphincteric areas, these react to any local or distant stimulus, be it either intrinsic or extrinsic in origin. The segments of the colon in response to viscero-motor, viscero-sensorial, and viscero-secretory stimuli react by annular spastic contractions in certain zones and thus modify the normal func-

tional activity of the large intestine. The function of these segments will be briefly considered.

(*A*) *Sphincter of Varolio.*—Due to its anatomical structure (Rutherford and Balli) the so-called ileocecal valve has the function of a genuine sphincter. In fact, the oblique fibers, which I have described, must have an influence on the function of this portion of the intestine. After the relaxation of the circular fibers of the sphincter, the passage of contrast substance through the sphincter is due not only to intestinal peristalsis, but also to the contractions of the oblique fibers. The sphincter of Varolio viewed from this standpoint has the function of regulating the passage of chyme from the ileum to the large intestine and impeding its reflux.

(*B*) *Sphincter of Busi.*—According to Busi, this sphincter does not generally contract as soon as the injected material has reached the cecum, but at a later time. It seems to have the function, in the beginning, of facilitating the absorption of material by the cecum, after which the contractions diminish.

(*C*) *Sphincter of Hirsch.*—According to Hirsch, the cecocolic tract acts to prolong the retention of the intestinal contents until cecal digestion and absorption are complete. He believes that the contraction and relaxation of this area of the intestine, analogous to that which happens in the pylorus, is regulated by chemical action.

(*D*) *Sphincter of Cannon-Boehm.*—There are supposed to be two distinct points of activity in this region, both subject to the influence of the nervous system (sympathetic and parasympathetic) which, however, can be stimulated synchronously, giving rise to movements which are well co-ordinated and produce colonic evacuation, or else react antagonistically, with the resultant stasis (ascending colon type).

One area, according to Arendt, shows a contracture formation, while the other area shows a short wave movement with antiperistalsis. The fine nerve connections among the inferior intestinal nerves, the renal nerves, and the pelvic nerves would explain the frequent propagation of stimuli from one organ to another. The contracture phenomena may show itself with or without enteric disturbances or, as a result of such lesions, as gastric ulcer, duodenal ulcer, and renal colic. There are no physiological data concerning the other sphincters.

Sphincters of the Colon in Relation to Some Abdominal Diseases.—Spasm of the sphincteric areas in the colon may be intrinsic or extrinsic in origin. I intend to consider briefly certain conditions, which can, at a distance, produce sphincteric spasm.

(1) *Dolichocolia.*—This abnormally long colon may be acquired or congenital. Some believe it to be congenital, but aggravated by the stasis and associated inflammatory changes. It is sufficient to state that following inflammatory changes with stasis and meteorism, spastic syndromes usually are found in those parts which show altered neuromuscular function and an inflamed mucosa. This does not mean that other areas of the colon cannot be involved. In cases of dolichocolia, I have observed spasm of the Payr-Strauss sphincter (splenic flexure).

(2) *Stypsis.*—The study of the various forms of constipation is very interesting even from the standpoint of differential diagnosis which can be made, radiologically, between true functional constipation and constipation due to extra-intestinal causes.

Radiological research in adults (Balli) and infants (Rossi) of the various forms of stypsis, as I pointed out at the Stockholm Congress, is of importance in the study of such conditions. I have encountered spasm of the Balli sphincter and the sphincter of Hirsch in cases of constipation; in others I have noted only a spasm of the former, and, in still others, spasm of Moutier's sphincter and the sphincter of Rossi.

(3) *Colitis and Pericolitis.*—The chapter on these lesions is of the most complex

of all abdominal pathology. The various types due to diverse causes give similar radiographic pictures. There are also simple irritative states which give etiological radiographic findings more or less similar to each other or altogether identical. In these forms, the spasm involves that portion of the colon which is affected by the morbid process. The corresponding sphincter in this area may become involved. Thus, the same may be said for pericolitis, in which for example, due to the presence of Jackson's membrane, there can be a spasm of Hirsch's sphincter (Hirsch and Balli) or a spasm of Busi's sphincter (Balli).

(4) *Diverticulosis and Diverticulitis.*—The study of diverticulosis and diverticulitis has assumed, in these past years, a great importance especially to the radiologist. The discussion concerning the sphincteric reaction in cases of diverticulitis, which we have been able to find reported in the literature, is not generally dwelt upon by the authors who describe abscesses, fistulæ, and peritonitis following the perforation of the diverticuli; or pericolitis, which, stenosing the intestine, may simulate constrictions due to neoplasms. Only now and then is there any reference to the phenomenon of spasm which is not infrequent in diverticulitis. Busi states: "The special spastic aspect of a segment of colon resembling an accordion, which simulates very much a dilated segment of small intestine with its valulæ conniventes, can often be found in diverticulitis and can even be considered characteristic."

Radiographically, it is simple to differentiate segments of colon-affected spasms due to multiple diverticulitis and from spasm due to extra-intestinal or other intestinal lesions. The presence of diverticuli, which are evidenced by small rounded shadows, grouped together or isolated, gives a characteristic radiographic picture which is unmodified even in the presence of spasm. The diverticuli in the spastic area stand out sharp and clear. In the majority of cases, the diverticuli are disseminated in large segments of colon and may produce a variety of spasms of the intestinal wall and in more than one sphincter at the same time.

(5) *Tuberculosis of the Colon.*—It is well known that the radiological findings of tuberculosis of the cecum show fixed deformities as a result of tubercular infiltration of the wall, and alterations in the cecum due to the more or less precocious emptying (Stierlin's phenomenon). However, in these forms, the spasm of Hirsch's sphincter (Piccinino) is also found.

In cases of ileocecal tuberculosis and in other tubercular conditions of the ileum, one can see spasms of Hirsch's sphincter (Hirsch, Balli, etc.).

(6) *Chronic Appendicitis.*—It is well known that the diagnosis of chronic appendicitis is given by numerous signs recognized by the radiologists.

In this regard Piccinino, Guidotti, and Perotti's research is quite interesting. According to Piccinino, "Besides the presence of gas in the terminal loops of small bowel, dilatations, hyperperistalsis of the terminal loop and disturbances of motility, inconstant ileal stasis, cecal stasis, initial accelerations of the motility of the colon, with successive delay in emptying, etc., there can be found deformities and spasms of the cecum and persistent contractions of the sphincter of Busi, together with a spasm of sphincter of Varolio. These findings are considered important for the diagnosis of chronic appendicitis."

Hirsch, some time ago, demonstrated spasm in the sphincter which bears his name, in cases of chronic appendicitis, and Busi, George and Leonard, and the writer also noted this finding on numerous occasions; the same applies to cases of appendicitis with adhesions of the right adnexa.

In appendicitis with dense adhesions, spasms of the sphincters of Busi, Hirsch, and Cannon are found simultaneously (Oppenheimer, Balli) and associated spasms of the ileocecal valve in patients in whom a diagnosis of chronic appendicitis was justified, have been noted by Labroche, Brodin, Bonneau, and Carrie.

Guidotti and Perotti (of my school) have studied the state of the colonic sphincters in chronic appendicitis and have found that there is a great accentuation of hyperexcitability of the colon in this disease, and, because of this, the sphincters show a disposition to spasm. They have found Busi's sphincter energetically contracted in all cases of chronic appendicitis which were studied; while the sphincter of Cannon reacted in only half the cases, the contraction being slighter, less diffuse, and limited to the right. The authors state that in one case of appendicitis the sphincter of Busi was contracted to such a degree that the cecum was not filled at all. At the operation, a few days after the radiological examination and 40 days after the acute appendiceal attack, a perforated appendix was found, with a small abscess circumscribed by peritoneal adhesions.

According to these authors, frequent spastic contractions of the sphincters of Hirsch, Payr-Strauss, Balli, and Moutier, and to a lesser extent Rossi's sphincter, may be demonstrated in these cases. The sphincter of Payr-Strauss has shown special sensitivity to contraction, yet this sphincter is located just below the splenic flexura.

(7) *Duodenal Ulcer.*—Sphincteric spasms together with pylorospasm may be found in cases of duodenal ulcer.

(8) *Cholecystitis.*—Guidotti and Perotti studying appendicitis and cholecystitis, by means of barium enemas, point out that credit is due the radiologists for revealing the complicated autonomy of the digestive tube and the existence of the various reflexes (viscero-sensorial, viscero-motor). The latter are especially important in our work because of the either increased or decreased motility of the colon that one finds in chronic cholecystitis.

According to these authors, the autoregulating mechanism of the digestive tube can be altered even by lesions of the gall bladder, with functional changes in the contractility of the various segments of the colon. From their study it is apparent that in chronic cholecystitis there is a state of hyperexcitability of the colon as demonstrated by the spastic contractions of various sphincters, induced by means of a tepid barium enema administered with low pressure.

The sphincters of Hirsch, Payr-Strauss, Balli, Moutier, and Rossi appeared to these authors contracted in an equal percentage of cases of appendicitis and cholecystitis (nine out of twelve cases).

Moreover, in some cases of cholecystitis the ascending colon and cecum have shown a diffuse haustration without a true spasm of Busi and Hirsch's sphincters.

Biedermann has demonstrated in cases of cholecystitis with stones a spasm of Cannon's sphincter, and a similar case has come to the attention of the writer.

CONCLUSION

1. The sphincters of the colon, the study of which is the work primarily of the radiologists, may from an anatomic standpoint, be divided into organic and functional types.

2. These sphincters have a notable physiologic importance because they play a part in the complex mechanism of the digestive tract.

3. These sphincters react by spastic contractures to lesions of the various segments of the colon and other abdominal viscera.

4. These sphincters do not necessarily act in the same way with a particular lesion, nor does the same sphincter always react to the same lesion.

5. The sphincteric spasm is not, therefore, a pathognomonic sign of an abdominal lesion, but is only a general sign of irritation.

BIBLIOGRAPHY

1. Aloi: Monitore Zoologico Italiano, 1932.
2. Balli: Atti del II Congresso Internazionale di Radiologia di Stocolma, 1928.
3. Busi: Bolletino scienze meche, 1924.
4. Hirsch: Atti del II Congresso Internazionale di Radiologia di Stocolma, 1928.
Idem: The Cecocolic Sphincteric Tract. Med. Jour. and Rec., **119,** 541-549, June 4, 1924.
Idem: Der Zöko-Kolische Sphinktertrakt. Fortschr. a. d. Geb. d. Röntgenstrahlen, **32,** 1924.
5. Perotti and Guidotti: Radiologia a Fisica Medica, Vol. 1.
6. Piccinino: Atti del Congresso di Radiologia Medica, Parma, 1932.

CONSIDERATION OF POLYCYTHEMIA AND GRENZ-RAY THERAPY[1]

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DIAGNOSIS AND ETIOLOGY

THE etiology of polycythemia is still a problem, the solution of which has not as yet been found. The standard texts and the literature consider polycythemia under two headings: (1) a pathological physiological response to tissue needs resulting from some peculiarities in the environment, and (2) a disease entity of unknown etiology and fatal termination.

Physiological polycythemia or erythrocytosis may be transient or permanent, trivial or grave, relative or absolute. It may result from a loss of tissue fluids, such as occur in cholera and profuse diarrheas. It may be due to localized concentration of red blood cells in the circulatory channels. It also occurs as a secondary bone marrow hyperplasia due to such conditions as high altitudes. When there are violent and abrupt transactions from sea level to high altitudes, cardio-vascular embarrassment occurs with increased production of red blood cells to compensate for the reduced oxygen tension of the atmosphere. Other circumstances, such as hot and cold baths, massage, violent exercise, and some drugs, following digestion, blood regeneration, vomiting, sweating, removal of exudates, myxedema, acute yellow atrophy, are followed by physiological polycythemia. Conditions of circulatory stasis as they occur in organic heart disease, as, emphysema, stenotic dyspnea, congenital diseases of the heart of the cyanotic type, also are frequently associated with polycythemia.

Under the names of erythremia, erythrocytosis, Vaquez's disease, Osler's disease, polycythemia rubra, and polycythemia vera, polycythemia has been considered as a disease entity. Erythremia is a slowly and intermittently progressive, ultimately fatal disease characterized by erythrocytosis, splenomegaly, and a peculiar red cyanosis with a greatly increased blood volume, hyperviscosity of the blood, a shortened coagulation time, and sometimes hepatomegaly. The etiology is definitely unknown, but familial and congenital relationships have been considered. Reznikoff, Foot, and Bethea reported, in 1935, 135 patients with polycythemia, of whom 48 per cent were Jews born in eastern Europe. This interesting etiological relationship is further brought out by the fact that there is an association between polycythemia and Buerger's disease. Brown, Allen, and Mahoner analyzed 100 polycythemic cases and found that 25 had symptoms and lesions characteristically found in thrombo-angiitis obliterans, and one case was definitely diagnosed as that disease. The pathological histology found in blood vessels of the bone marrow is similar to that found in cases of Buerger's disease.

Erythrocytosis, splenomegaly, and the peculiar red cyanosis of the skin represent the triad of symptoms found in the majority of cases of polycythemia vera. Sometimes bilateral exophthalmos, blurring of vision, dizziness and headaches, gastrointestinal disturbances, anorexia, vomiting, constipation, hemorrhages, and cardiovascular symptoms are prominent in the history. The kidneys show albumin and casts, and sometimes moderately raised, or even high, blood pressure is present. It is considered by Brown and Giffen that renal function is slightly impaired. The hematology of polycythemia always shows an absolute increase in blood volume. The red blood cells are increased up to fifteen million and the hemoglobin may go as high as from 165 to 220 per cent, with a low color index. The specific gravity of the blood is always high but since the serum

[1] This is one of a series of papers contributed by friends and former pupils of I. Seth Hirsch, M.D.

is normal, this is due entirely to the increased number of the red blood cells. The fragility of the red blood cells may be normal or slightly diminished. The oxygen-combining power of the venous blood is normal, but the venous blood seems to be rich in oxygen. The heart may be enlarged sometimes; the circulation is sluggish but the heart volume output is normal. The red blood cells show anisocytosis and polychromatophilia, and nucleated red cells and myelocytes are found Minot and Buckman showed definite abnormality in the blood smear by the demonstration in all cases of immature forms of red blood cells.

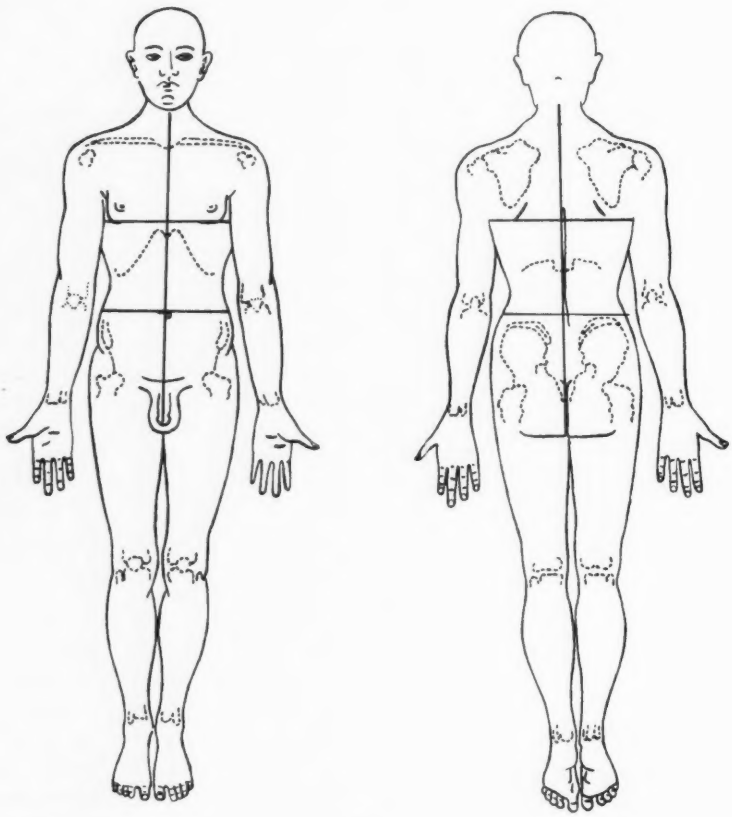

Fig. 1.

Pathology.—Reznikoff, Foot, and Bethea reported that bone marrow vessels show capillary thickening, probably fibrosis, subintimal and adventitial fibrosis of the capillaries, arterioles, and arteries in cases of polycythemia vera. Controls in cases of secondary polycythemia, aplastic anemia, and aleukemic leukosis were all normal and arteriosclerotic and arteriolarsclerotic controls all showed medial and adventitial fibrosis only.

In the polycythemia cases, occasional thrombosis of small arteries was found, fresh and organized. This study demonstrated that in polycythemia vera there is a definite pathological picture of the capillary and arteriolar blood vessels supplying the bone marrow, but whether this is due to the disease process of polycythemia or causative of the disease process is not dis-

cussed in the literature. However, it is considered by Minot that the pathological processes in the small vessels cause a condition of reduced oxygen tension in the bone marrow with a compensatory or overcompensatory hyperplasia of the bone marrow with the production of an increased number of blood cells.

A mild, early case of polycythemia showed acute inflammatory and necrotic lesions along the course of the blood vessels, characterized by cellular exudates which formed perivascular "cuffs" and some capillaries showed thickening in the midst of pronounced inflammatory lesions. The exudates consisted of lymphocytes and monocytes caught in the migratory, elongated phase. There were a few neutrophiles which rarely formed miliary abscesses. Brown and Giffen studied the capillary morphology in these cases and found that there was engorgement of the venous limb and collecting venule. The arteriolar segment showed relative contraction or a smaller lumen and occasional engorgement. These authors concluded that the tortuosity depended on the age of the patient and not on the blood volume. They found 65 open capillaries per square mm. of skin surface, whereas the normal is from 15 to 40. They also demonstrated that the capillaries came back to normal with reduction of the blood volume. There appeared to be, in polycythemia, a complete utilization of all the capillaries. Return to normal occurred when the blood volume was reduced from 166 c.c. per kilogram of body weight to 110 c.c. Brown and Giffen consider that the thrombotic processes result from an increase in the viscosity and changes in the vessel wall, and changes in the coagulation factors of the blood.

Changes in capillary walls may be due to strain, metabolic disturbances, and other factors. It is, therefore, difficult to conclude from this study whether the changes in the capillaries, which may be due to pre-existent disease, are the cause of polycythemia or the direct result of the pathological processes in polycythemia and of the changes in the blood due to polycythemia. The clinical sequence of events in polycythemia vera seems to follow the following pattern.

A disease process of unknown etiology causes an increase in the number of red blood cells of the circulating blood. The same disease process, or the changes in the blood due to the disease process, causes inflammatory changes in the capillaries, arterioles, and arteries of the bone marrow. These changes are so similar to those found in thrombo-angiitis obliterans of Buerger that the disease has been considered thrombo-angiitis obliterans of the bone marrow. As the disease becomes more chronic, there occurs fibrosis with or without increase in the red blood cells, with or without remission. The permanency of fibrotic changes in the blood vessels refutes the possibility of a remission in erythrocytosis since the fibrosis should act as a permanent impediment to transmission of oxygen to the cells of the bone marrow and cause permanent reduced oxygen tension in the bone marrow. Remissions are not explainable, therefore, and erythrocytosis does not always occur in polycythemia. After adequate venesection, remissions are induced; yet phlebotomy can only reduce the number of oxygen carriers of the bone marrow and further reduce the oxygen tension, but the bone marrow ceases to produce an abnormal erythrocytosis. It is possible that the fundamental but unknown cause of polycythemia may be the cause of tissue hyperactivity of the erythrocyte-producing cells of the bone marrow and that this tissue hyperactivity is the cause of chronic stimulation of the inflammatory reactions, both acute and chronic, in the bone marrow vessels.

Treatment.—The accepted treatment of polycythemia vera comes under three classifications:

(1) Venesection for the purpose of reducing the blood volume. Stephens and Kaltreider removed from 1,000 to 3,000 c.c. of blood in a few days by repeated phlebotomies of from 200 to 600

c.c. of blood and found that hematological and clinical remissions, varying in duration from two months to two years, occurred. There was no variation in the normal range of reticulocyte reactions. These authors believed that a removal of iron and other blood-building materials which are stored with the hemolytic action of phenyl hydrazine, was responsible. Dameshek and Henstell subjected their patients to venesection of 500 c.c. of blood twice weekly till the hemoglobin fell to between 70 and 80 per cent and the red blood cells to about five millions. Their patients were limited to a low iron diet containing less than 6 mgm. of iron per day. They remained symptom-free from six to nine months and rising values were controlled by a few venesections.

(2) By hemolyzing the excess blood cells with some drugs such as benzene, phenyl hydrazine, and acetyl phenyl hydrazine. Experimentally, Waltner and Waltner showed that cobalt given to animals causes polycythemia, and Mascherpa showed an increased activity in the bone marrow in these animals. Barron and Barron believed that cobalt polycythemia is due to the inhibition by cobalt of the respiratory function of the immature red blood cells. These cells are thrown into the general circulation as mature non-respiring cells, being replaced in the bone marrow by new cells. In animals, the administration of ascorbic acid with cobalt prevented the formation of polycythemia and after cobalt administration, polycythemia was reduced to normal levels. Kandel and Leroy administered ascorbic acid to polycythemic patients and found that it had no effect, either good or bad, on the hemoglobin and red blood cell levels in these patients, especially in the prevention of a return to polycythemic values.

(3) Milani, in 1929, and Pack and Craver, in 1930, reported good results with radiation. In 34 instances, Sgalitzer radiated the whole body with fair results, but no permanent remissions. He applied 25 r for 20 minutes to the whole body for six successive days, followed by a weekly interval of rest, with the course repeated until the red cell count fell to normal, noting the fall in leukocytes and stopping the treatment when they fell to 3,000 per cubic millimeter.

It can be seen from this short review of the subject that although there has been advancement in the knowledge of the pathology and experimental consideration of polycythemia, we have not reached a knowledge of the fundamental cause or a satisfactory treatment in polycythemia vera. It is, therefore, of interest to point out the fact that Grenz rays have produced noteworthy clinical and hematological improvements of long duration and to attempt an explanation for the results obtained in these studies.

It is beyond the scope of this review to go into the technical, physical, biological, and physiological aspects of Grenz rays; it is sufficient for our purpose to be brief in describing their nature.

Grenz Rays.—These rays are electromagnetic oscillations of about two Ångström units which produce characteristic biological and clinical manifestations. Absorption of Grenz rays is almost complete in the skin—88 per cent are absorbed in three mm. of skin and only 12 per cent reach subcutaneous tissues in this instance; while more than 90 per cent of roentgen rays pass through such a layer of skin.

It seems highly probable that the kinetic changes in atoms and molecules are more or less the outcome of radiation, though here we deal with orders of magnitude lying far below those which the biologist explores so freely with his microscope (9). Still, if we may regard such transformations as the antecedents and causes of chemical phenomena, and thus of vital activities, it is evident that to radiation there must be assigned, in theory at least, a fundamental influence on the very life process itself; an influence which often is not limited to a single organ but may embrace an entire system.

Interesting and valuable as the Grenz ray is in dermatology, its action upon

internal diseases is even more striking. It might appear irrational at first glance to attempt the treatment of internal diseases by influencing the skin, but a few theoretical considerations will serve to clarify the situation.

More complicated, but for that reason much more interesting, are those processes which take place in the skin on the one hand, and the autonomous nervous system on the other. Here belongs the projection of internal phenomena upon the skin surface, of which only Head's zones and the condition known as "goose flesh" need be mentioned for our present purpose. It is but natural that reactions taking place in the opposite direction, as when events in the skin are reflected by the internal organs, should be harder to investigate, and it is just this difficulty that has delayed correct interpretation of the consequences attendant upon severe and extensive burns of the skin until very recent times. These include diarrhea and intestinal hemorrhage, which have been ascribed to toxic products absorbed into the circulation from the burned surface, notwithstanding the fact that such symptoms never accompany other sorts of injury with equal tissue destruction. Nor has it ever been explained why analogous effects are not produced in all the other internal organs.

On the other hand, an alternative conception which regards these consequences as an outcome of the autonomic connection between skin and splanchnic system, is supported by a whole series of observations. Perhaps the most important of these is a fall in the leukocyte count. The articles of Glasser, E. F. Muller, Petersen, Emden and Freundlich, and others contain a wealth of material relating to this phenomenon which is too important to be overlooked.

Irritation of the skin by light massage, intradermal injection of any material whatsoever (physiological salt solution or air), and so on, is followed after about ten minutes by a fall of some 20 to 30 per cent of the original leukocyte count; the number returns to normal, however, in the course of from 20 to 30 minutes. It is to be emphasized that this return is a characteristic feature of the drop, distinguishing it from persistent leukopenia. As the decrease of leukocytes in the skin is accompanied by a rise of those in the splanchnic system, transfer from the surface to the interior must take place, and the influence of the autonomic nervous system in this emigration is clearly suggested by the work of Emden and Freundlich. These investigators found that the fall did not occur in a sympathectomized limb, though it could be elicited as usual in the normal member of the opposite side.

As the skin and bone marrow are known to be connected through the autonomic nervous system, the treatment of polycythemia rubra was begun. The most effective therapy for this disorder is roentgen radiation of the long bones, as is well known, on the hypothesis that the function of the bone marrow can be thus inhibited and the production of erythrocytes thereby diminished. Well filtered hard rays are accordingly employed, in order that the greatest possible amount of energy may reach the marrow, and the results of this method are really excellent. But it has been shown that general radiation with Grenz rays will also influence favorably the course of this disease in the great majority of cases. It has been our experience in polycythemia, as in vitiligo and alopecia, that a decisive result is not to be expected from but one series of treatments and the physician should not be discouraged, therefore, if improvement does not set in even after the lapse of several weeks. The first evidence is generally symptomatic (amelioration of dizziness, headache, etc.), and not until later does the diminution in erythrocytes and hemoglobin begin to be demonstrable. Indeed, we have frequently seen a rise in the red cell count and the hemoglobin percentage, and in such cases have achieved complete success only after repetition of the series; in this way we were able

to mitigate the condition of a patient in whom Grenz-ray treatment had been discontinued prematurely because it was thought to be ineffective. It is just this possibility of "repetition until success is attained" that makes the Grenz ray preferable to the x-ray, in which recurrent treatment is definitely barred (Spiethoff). By repeating the series throughout the course of one or two years, we have ultimately relieved most of our patients. In the rather extensive literature dealing with polycythemia, remission has been reported only by Rosin and Weintraut, so far as the writer is aware, and it must, therefore, be an unusual occurrence. Hence, any assumption that alleviation occurred spontaneously in all our patients who were improved can be immediately dismissed; of a spontaneous cure we have been able to find no record.

An attempt to explain the manner in which the Grenz ray acts in this disease leads to interesting considerations. It is still a debatable question whether the excess red cells indicate increased production in the bone marrow or decreased destruction in the spleen. The former hypothesis, which underlies the treatment of polycythemia with x-rays, would appear to have been confirmed were it not that cases have been recorded in which roentgen radiation of the spleen alone has been more or less successful. Nevertheless, it is now generally conceded that radiation of the bone marrow is incomparably more efficacious. As the changes in bone marrow and blood are constant findings, whereas splenic enlargement may not be present, it seems logical to search for the seat of the disease in the marrow rather than in the spleen, and the more so because there is no relationship between the erythrocyte count and the size of the spleen; indeed, the fact that an enormous increase in the blood count may be associated with a small spleen, and *vice versa*, discourages all attempts to implicate the spleen itself. However, the disease has a number of other inconstant and puzzling features. So, for example, ulcerations in the gastrointestinal tract are not an uncommon accompaniment, and perhaps are more often encountered than the literature would suggest (von Bergmann's dysharmony of the vegetative system). Autopsy discloses no change common to all cases, and it is highly probable that the variable symptomatology is the outcome of functional disturbances in one common center, manifesting themselves now in this organ, now in that, according to its "tone" or "disposition."

Our experience with Grenz-ray therapy corroborates the assumption that polycythemia is associated with a functional disturbance in the autonomic nervous system, which normally should supervise and regulate all activities of the various organs. At the present time this is, in fact, the only possible explanation for the favorable results brought about by these rays, for it is inconceivable that any proportion of them worth mentioning should ever reach the bone marrow, their influence being confined, as it is, to the skin. Hence, the possibility that they work as the x-ray is supposed to act, may be denied. It may still be asked, however, if the Grenz rays may not directly destroy the erythrocytes in polycythemia. The latent period of several weeks which must elapse before any diminution appears in their number is against any such suggestion. Furthermore, the amount of blood actually radiated is insignificant, as the rays penetrate hardly more than three millimeters and the exposed field is but 15 cm. in diameter, while the exposure is brief and the amount of available energy relatively small; whence it follows that a minimal volume of blood is affected by comparatively little radiant energy.

Again, symptoms such as we are accustomed to associate with the destruction of red blood cells do not arise, or at least are not appreciable. One last question is, whether some sort of by-product, or a hormone, perhaps, may not be elaborated in the tissues or the blood, but so far we have seen no evidence to support the suggestion, though we have applied Grenz

rays over large areas in a varied assortment of diseases. Of course, some intermediate substance, something of the nature of histamine, might be formed which would affect the autonomic system secondarily, but it seems far more probable that the Grenz rays exert their regulatory effect on organ function through the skin and the autonomic nervous system, as has already been explained in the section on leukocyte drop and sympathectomy.

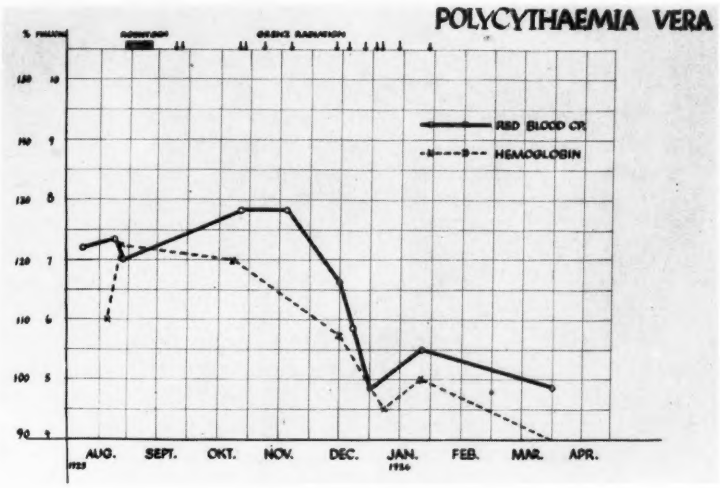

Fig. 2. Case 1.

Comparison of the treatment of polycythemia with x-rays or Grenz rays suggests certain interesting conclusions. Ludin's idea of influencing the bone marrow directly was plausible enough and appeared to be supported by the results. But though it may be safely assumed that in the case of the Grenz ray any such effect can be excluded, the results resemble those of deep therapy. This might be taken to mean that there are actually two distinct ways of affecting the function of the marrow, since the hard rays act directly upon it and the Grenz rays only indirectly. Whether this deduction be permissible cannot be decided at the present time. At any rate, the possibility remains that the hard rays themselves also have an indirect effect, which is exerted partly or wholly through the skin. Against this, however, may be set the fact that the results of treating the spleen with rays have been so unsatisfactory as to cause abandonment of the method; still, it must be borne in mind that in treating this organ a smaller field is generally employed than when the exposure is directed at the bones, so that the amount of irritation set up in the skin is correspondingly less. The total dose, too, is smaller. Skin absorption is negligible with hard rays in comparison with Grenz rays, though the difference may be offset to a certain degree by the relatively large doses of x-ray which are required to produce any effect upon polycythemia. It appears to be of significance, also, that only one or two treatments are administered when the spleen is exposed, whereas exposures to the bones are continued for a longer period, so that, as with nonspecific protein therapy, stimulation takes place repeatedly.

The following technic, based upon principles that are still valid, has been em-

ployed from the first. The surface of the trunk is divided into from eight to twelve fields—from four to six on the anterior and from four to six on the posterior aspect—so as to give a right and a left thoracic and a right and a left abdominal area, with corresponding fields on the dorsal surface. Assuming that, as with non-specific protein therapy, the stimulus would have to be repeated and the treatment, therefore, extended to cover a certain period of time, an area was treated daily, so that after the lapse of eight days each had had one radiation. In order to prolong the stimulation, the whole procedure was then repeated, except that now an interval of one day was interpolated between each two exposures. This procedure required 24 days, and one complete treatment of this sort was called a series. A series may be prolonged, if desired, by the inclusion of additional fields (hip, extremities, etc.). It is necessary in most cases to repeat such a series, and it has been our custom to allow an interval of from six to eight weeks before beginning the next one. There is not the slightest danger in repetition, for doses are employed which cause no demonstrable changes in the skin; even a definite pigmentation is seldom observed. Nor is there any objection to administering series after series, if the appropriate intervals be observed, for no damage has ever resulted in cases in which the proper technic has been followed. It seems scarcely necessary to say that the apparatus and the tube itself must be carefully calibrated, for failure or disagreeable consequences may attend faulty technic. Simple though the procedure may be in itself, it must be based upon scrupulously exact dosage, and must, therefore, be supervised by experienced operators.

The best means of measuring the output at the present time is the ionization method, for which a number of reliable instruments are available. The single dose which I have employed amounts, at 15 cm. focal skin distance and with a field about 15 cm. in diameter, to some 150 r; a half absorption value of from 0.0175 to 0.02 mm. in aluminum is to be preferred. In our series, five patients showed definite symptomatic and hematological improvement out of a total of seven, or approximately 71 per cent.

One case made no response to adequate treatment and we could find no explanation for the failure. The other case made no response either, but in this instance, general roentgen radiation had been given. It is usual to find the cases more resistant to Grenz-ray therapy if previously given such treatment.

Case 1. L. B., a 49-year-old houseman (Russian Jew), was admitted to the hospital on Aug. 10, 1925.

His family history was irrelevant. He had typhoid as a child, after which he was not ill for a long time. He smoked heavily. For three months he had had epigastric pressure and pyrosis, appearing every three or four days some three hours after meals. The pain also came at night and when the patient was hungry. It radiated downward to the left. There were severe eructations, loss in weight, constipation, headache, and dizziness. He noted that his face was turning a reddish-blue.

The patient was weak and somewhat undernourished. He presented the characteristic dusky, cherry-red complexion of polycythemia, with cyanotic nose and ears and injected conjunctivæ. The epigastrium was tender, the spleen just palpable. Other physical signs were irrelevant. Roentgen examination showed an irregular descent of the duodenal bulb, diagnosed by Dr. Gottlieb as duodenal ulcer. Blood pressure on Aug. 11, 1925, was 94/66; Sept. 22, 1925, 120/60. Marked traces of occult blood in the feces were found twice.

The patient could not be persuaded to take treatment regularly; therefore, consecutive observations were impossible and the treatment dragged along. After roentgen treatment in August and September, the red blood cells increased in number, reaching the peak in November. Only two Grenz-ray treatments had been given

in September. After the end of October, the patient was more regular in his visits. The arrows indicate the periods of treatment, but not each individual exposure

cramps. The family history was irrelevant. The patient had had pneumonia 33 years before, and a tremor of the hands for 30 years, a laparotomy 12 years

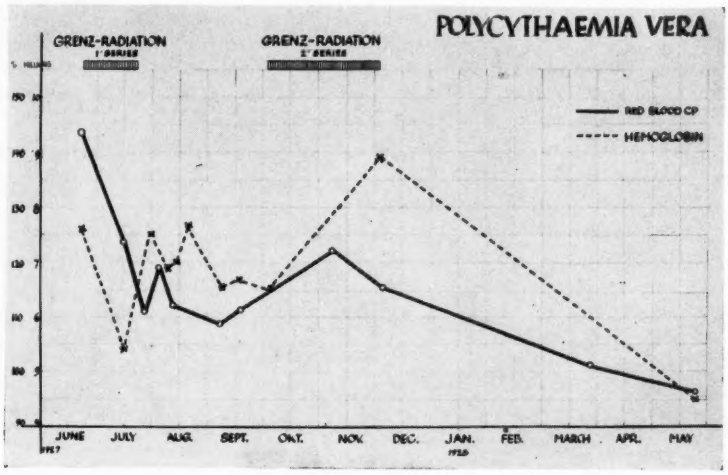

Fig. 3. Case 2.

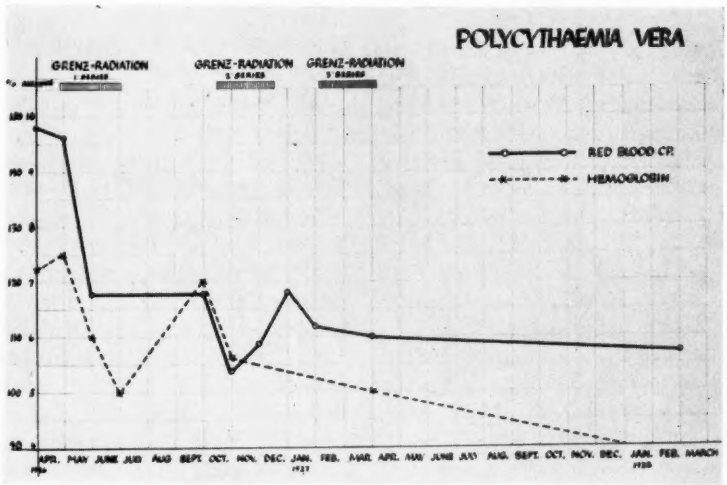

Fig. 4. Case 3.

(Fig. 2). The diagnosis was polycythemia and duodenal ulcer.

Case 2. P. B., a 68-year-old smith (Jewish), had complained of difficulty in evacuating, headache, and abdominal

previously, and for 11 years had been unable to work because of weakness. The man was somewhat undernourished, with a slightly blue face. He had no marked disabilities. The pupils were sluggish,

and the pharynx was red. His thorax was barrel-shaped and covered with a maculo-papular rash. Save for a few râles at the right base in the back, the lungs were negative. The heart was ash. The hemoglobin was 125 per cent; red blood cells, 9,400,000; white cells, 12,000. The polymorphonuclear leukocytes were 87 per cent; small lymphocytes, 6 per cent; normocytes, 2 per cent;

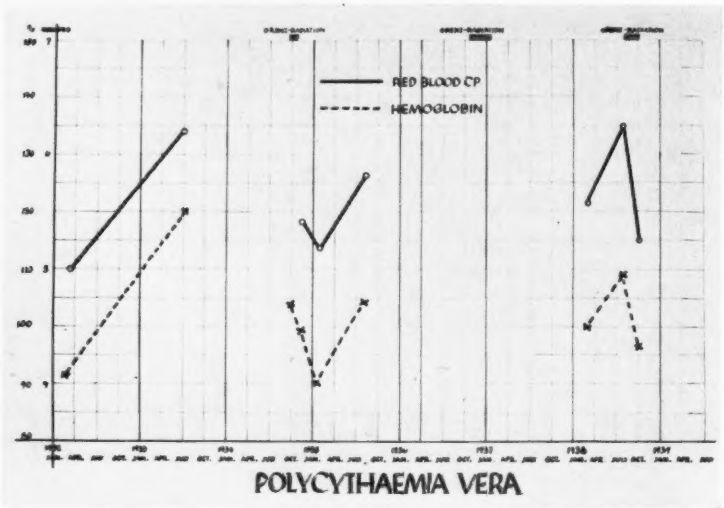

Fig. 5. Case 4.

slightly enlarged as was also shown on roentgen films. The electrograph was normal, and save for palpability of the spleen the abdomen was negative. The reflexes were increased. Hemoglobin was 124 per cent; the red blood cell count, 9,600,000; the leukocytes, 16,000.

Case 3. L. H. S., a 60-year-old Jewish physician, had been ill for seven or eight years. The family history was negative. The patient had had a duodenal ulcer for 15 years. His chief complaints at this time were headache after slight excitement, excitability, loss of self-control, insomnia, sluggishness of the bowels. He was easily fatigued. His left great toe had hurt him for nine months, but the pain had receded during the last three months. His blood pressure was 150/85. The spleen was markedly enlarged, its lower pole reaching the iliac crest. The lips, nose, and ears were blue, the hands red.

There was slight albuminuria with somewhat diminished chlorides and residual eosinophiles, 4 per cent, and basophiles, 1 per cent.

About seven years previously, he had had roentgen-ray treatment, starvation therapy, and fruit diet. After the last, the patient maintained that the red blood cells had dropped from 8,000,000 to 4,500,000, but for only eight months. The second series of treatments extended over a protracted period, the patient having been irregular in attendance. He died some years later from pneumonia.

Case 4. The patient, R. P. (Jewish), was seen in 1931, with a history of substernal pressure and pain radiating to the left arm. The physical examination was negative. The blood pressure was systolic, 132; diastolic, 84. X-ray examination of the heart showed normal diameters. Gastro-intestinal examination was negative. The impression at that time was that of coronary disease. The following week the patient had a definite attack of coronary thrombosis which necessitated confinement

to the Beth Israel Hospital for one month.

In February, 1932, the blood count showed the following results: hemoglobin, 91 per cent; red blood cells, 5,000,000; white blood cells, 8,100. In July, 1933, the spleen became palpable. The patient was considered to have a coronary thrombosis, after which a polycythemia vera developed. Figure 5 shows the effect of Grenz rays on the blood count.

Case 5. Mrs. T., a middle-aged Jewish housewife, had been known to have polycythemia vera since 1927. She had been treated with venesections and diet, with poor results. In 1930, she started Grenz-ray therapy and at that time was suffering from dizziness and headaches, and was unable to walk. She had the characteristic red cyanosis and complained of gastro-intestinal symptoms characteristic of ulcer. After Grenz-ray therapy, all symptoms were greatly improved and Figure 6 shows the effect on the red blood cells. She died in 1935. Multiple abdominal and pulmonary thromboses were found, at postmortem examination, which were considered to be the results of polycythemia vera, in spite of the improvement shown symptomatically and hematologically.

The course of the disease and effect of the Grenz rays can be seen from the curves presented (Figs. 2–6).[2] Immediately after treatment there was no change found in the clinical picture. Both subjective and objective improvement set in after three or four weeks. The face grew noticeably paler, the headaches and dizziness gradually vanished. Together with this, the erythrocytes and hemoglobin dropped. There was virtually no effect on the leukocytes.

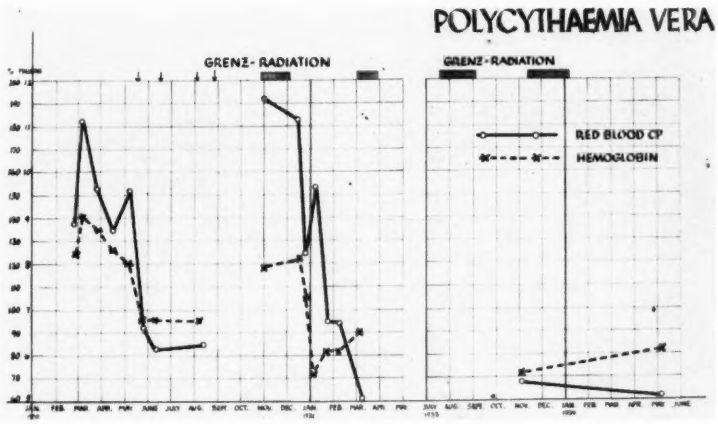

Fig. 6. Case 5.

BIBLIOGRAPHY

(1) WALTNER, KLARA, and WALTNER, KARL: Klin. Wchnschr., **8,** 313, Feb. 12, 1929.

(2) MASCHERPA, P.: Arch. ital. de biol., **82,** 112–120, 1930.

(3) BARRON, A. G., and BARRON, E. S. G.: Mechanism of Cobalt Polycythemia: Effect of Ascorbic Acid. Proc. Soc. Exper. Biol. and Med., **35,** 407–409, December, 1936.

(4) KANDEL, E. V., and LEROY, G. V.: Note on the Lack of Hemoregulatory Effect of Ascorbic Acid on Patients with Polycythemia Vera. Am. Jour. Med. Sci., **196,** 392–395, September, 1938.

(5) MILANI, G.: Roentgen Treatment of Vaquez's Disease. Jour. Am. Med. Assn., **93,** 1205–1208, Oct. 19, 1929.

(6) PACK, G. T., and CRAVER, L. F.: Radiation Therapy of Polycythemia Vera. Am. Jour. Med. Sci., **180,** 609–617, November, 1930.

(7) SGALITZER, M.: Total Irradiation with

[2] A recent case showed the following figures: Feb. 26, 1939, erythrocytes, 8,000,000; hemoglobin, 130 per cent; Aug. 12, 1939, erythrocytes, 4,600,000; hemoglobin, 100 per cent.

Roentgen Rays in Polycythemia Cases. Wien. klin. Wchnschr., **48,** 675–677, May 18, 1935.

(8) BUCKY, G.: Grenz-ray Therapy. The Macmillan Co., New York City, 1929.

(9) Idem: Grenz-ray Therapy of Internal Diseases. Med. Rec., **142,** 1935.

(10) BUCKY, G., and FREUND, E.: Die Beeinflussung der Polycythaemia rubra durch Grenzstrahlen. München. med. Wchnschr., **75,** 1405–1408, Aug. 17, 1928.

(11) REZNIKOFF, P., FOOT, N. C., and BETHEA, J. M.: Etiologic and Pathologic Factors in Polycythemia Vera. Am. Jour. Med. Sci., **189,** 753–759, June, 1935.

(12) BROWN, G., ALLEN, E. V., and MAHONER, H. R.: Arch. Intern. Med., **54,** 436, 1934.

(13) MINOT, G. R., and BUCKMAN, T. E.: Erythemia (Polycythemia Rubra Vera). Am. Jour. Med. Sci., **166,** 469, 1923.

(14) BROWN, G. E., and GIFFEN, H. Z.: Vascular Changes in Cases of Polycythemia Vera. Am. Jour. Med. Sci., **171,** 157, 1926.

(15) KRACKE, R. R., and GARVER, H. E.: Diseases of Blood and Atlas of Hematology. Lippincott, Philadelphia, 1937.

(16) BROWN, G. E., and GIFFEN, H. Z.: Studies of Capillaries and Blood Volume in Polycythemia Vera. Am. Jour. Med. Sci., **166,** 489, 1923.

(17) STEPHENS, D. J., and KALTREIDER, N. L.: Therapeutic Effect of Venesection in Polycythemia Vera. Ann. Int. Med., **10,** 1565–1581, April, 1937.

(18) DAMESHEK, W., and HENSTELL, H. H.: Treatment of Polycythemia Vera by the Production of a Chronic Iron Deficiency. Jour. Clin. Invest., **16,** 683, 1937.

(19) ELWARD, JOSEPH F.: Radiation Therapy. RADIOLOGY, **26,** 86–98, January, 1936.

(20) BAMBERG, H. L., and KRÖKER, P.: Treatment of Lupus with Grenz Rays. München. med. Wchnschr., **84,** 569–571, April 9, 1937.

THE IMPORTANCE OF SACRO-ILIAC CHANGES IN THE EARLY DIAGNOSIS OF ANKYLOSING SPONDYLARTHRITIS[1]

MARIE-STRÜMPELL-BECHTEREW DISEASE

By JACQUES FORESTIER, M.D. (Paris), F.A.C.R. (Hon.), *Aix-les-Bains, France*

I

AMONG the chronic diseases of the spine, ankylosing spondylarthritis[2] is one of the most crippling, since its natural result is the complete bony ankylosis of the whole spine. It is a systemic disease which, in its advanced phases, can involve the main joints of the limbs (*spondylose rhizomélique* of Pierre Marie).

The nature of the disease is not yet absolutely determined but it is generally accepted that it starts as an atrophic arthritis of the joints between the facets of the vertebræ (apophyseal joints) which, after an inflammatory stage, become involved by bony ankylosis. Secondarily, there are ossifications of the adjoining ligaments in the ligaments flava, in the anterior and posterior vertebral ligaments, resulting in the classical picture, in the roentgenograms of the bamboo-spine.

The clinical picture in the early stages of the disease is very confusing, which is most regrettable since it is proven that the disease is much easier controlled by treatment at this period, before the advent of the ossifications, than later on when the stiffness of the spine is irremediable. The most striking features of the early period are vague and diffused low back pains, sometimes located in the vicinity of the sacro-iliac joints. Not infrequently an acute attack of sciatica is the only clinical incident which marks the onset of the disease. Still more confusing are the cases associated with radiating pains around the chest or the abdomen which are often mistaken for pleurodynia, kidney disease, intestinal disturbances, uterine retroversion, etc.

The importance of an early diagnosis of the disease cannot be too greatly emphasized. As long as only inflammatory changes are present in the intervertebral joints and even when some purely fibrous adhesions have taken place, ankylosis of the spine can be avoided by effective treatment which has been established in the past years and which can, in most cases, check the evolution of the disease before ankylosis has taken place. But it must be admitted to-day that such an early diagnosis is missed not only by practitioners but even by qualified specialists. It is our experience that over 80 per cent of the cases of ankylosing spondylarthritis that have come to our observation in the past three years, before the beginning ossifications in the spine, have not been diagnosed as such. Most of them were labelled sciatica, muscular rheumatism, lumbago, colitis, pyelitis, etc.

A short survey of the principal features of the disease at its early period will establish the basis of the problem. The age of the patients varies between 20 and 35, most of them, after careful questioning, relating the onset of the first mild symptoms to have occurred between the ages of 18 and 25 years. Male patients are observed almost exclusively, the disease among women being a rarity.

In 60 per cent of the cases, a history of past genito-urinary ailment is reported, not always gonococcal, sometimes due to *Bacillus coli* or any other infectious germ. It is quite suggestive that these patients are relieved of their lumbar pains by the reclining position in the first part of the night, but are frequently obliged to get up several times in the second part of the night

[1] This is one of a series of papers contributed by friends and former pupils of I. Seth Hirsch, M.D.

[2] The abbreviation "A. S." will be used for "ankylosing spondylarthritis" in the course of this paper.

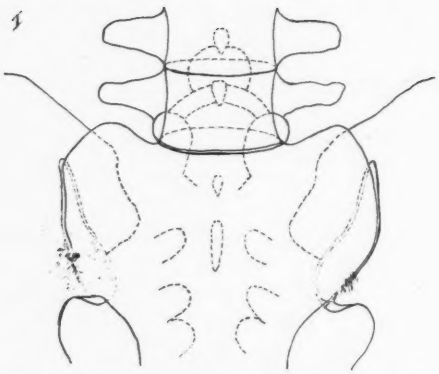

Fig. 1. Sacro-iliac changes in ankylosing spondylarthritis. First stage, marginal decalcification.

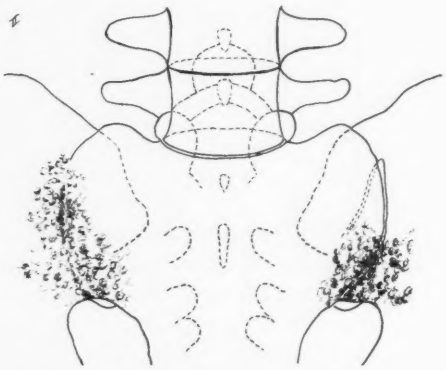

Fig. 2. Second stage, picnotic formation. A large area of bone, both on the sacrum and on the ilium, shows a mottled appearance with decalcification and hypercalcification.

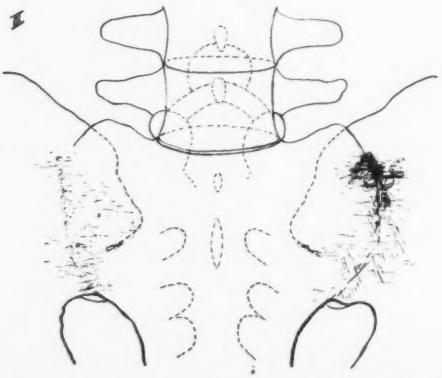

Fig. 3. Third stage, terminal stage. Total synostosis with fibrillary ossification and areas of osteosclerosis.

to relieve the muscular spasm in their spinal muscles. The value of blood tests to prove that an inflammatory condition is present is almost as important as in rheumatoid arthritis. Over 80 per cent of these patients show an accelerated sedimentation rate, which we check in France by the resorcine flocculation test (Vernes), which gives almost constantly high figures in the active part of the evolution of ankylosing spondylarthritis.

For many years, the roentgenographic examination has not been very helpful in the early diagnosis of A. S., since clinical observation has proven that a long period of time could elapse before some degree of ossification (bridging) appears between the vertebræ. Not only are these changes not visible roentgenologically in the inflammatory period of the disease, but fibrous ankylosing may have taken place and stiffened a part of the spine without any apparent changes other than a mild diffuse general osteoporosis of the bony structures of the pelvis and the spine. The intervertebral discs are generally normal in height and the contours of the vertebræ are normal both of the bodies and of the various processes.

Some authors have claimed that early changes could be detected on x-ray films with a proper technic (oblique views) in the joint spaces of the articulations—thinning of the joint spaces, decalcification of the articular processes. However, experience shows that the interpretation of these changes is often subject to criticism. It has been reported that the facet joints may appear normal even when ossifications had already appeared between the bodies of the vertebræ.

We have found, during recent years, that in opposition to these doubtful vertebral signs that *the roentgenographic changes in the sacro-iliac joints are of enormous value in the early diagnosis of A. S.* It is our aim to prove that the association of some clinical signs in the spine and especially in the lumbar region (pain, stiffness, lumbosciatica, etc.), with some definite roentgenographic signs in the sacro-iliac joints,

are pathognomonic of A. S. To demonstrate the correctness of our views, we shall discuss the following points:

(*A*) Are sacro-iliac joints always involved in the course of A. S.?

(*B*) Are sacro-iliac joints involved *previous* to the vertebral changes?

(*C*) What are the typical roentgenographic changes which sacro-iliac joints undergo at the beginning of A. S.?

II

Sacro-iliac Joints in the Course of Ankylosing Spondylarthritis.—Though Pierre Marie and Léri had reported in their first description of the pathologic changes of the disease that the sacro-iliac joints had undergone a bony ankylosis, it is only in the last decade that the roentgenologists have noticed that this fusion appeared to be associated with the well-known appearance of bamboo-spine. In a review of the modern literature of A. S., we have found this fact mentioned by Krebs and Vontz (2), Swaim and Kuhns (3), and others. In 1936, G. Scott (4), reporting 110 cases of what he called "spondylitis adolescens," found in all cases without exception either a synostosis of these joints or some abnormalities in their roentgenographic appearance. C. W. Buckley, in 1935 (5), in a wide survey of more than a hundred cases, stated that in the majority he had found a complete bony ankylosis of the sacro-iliac joints: in one case only these joints appeared to be normal. In nine cases, some gross changes had taken place but the joints were not fused. Both Scott and Buckley reported a few cases in which these sacro-iliac changes had been detected at an early period of the evolution of the disease and previous to any changes in the roentgenologic aspect of the joints of the facets. Personally reviewing 153 cases of A. S. which came under our observation at any period of their disease, we found only two cases in which the sacro-iliac joints did not appear to be roentgenologically involved. The first case was that of a middle-aged man complaining of pains in his knees, without any vertebral symptoms, but we found some stiffness in his neck and the roentgenogram revealed two ossifications (bridging) uniting some cervical vertebræ. The sacro-iliac joints and the lumbar vertebræ were normal; the sedimentation rate was also normal. We are not certain that this case was a true A. S.

The second case is a man, 60 years of age, affected with diabetes for about ten years, who started, about the same time, to complain of lumbago and pains radiating from the pelvic girdle. When we examined him, his spine was incompletely ankylosed in its three segments and both hips were very much impaired in their movements. The sedimentation rate was 26 for the first hour (Westergren) and the resorcine flocculation test was also abnormal. The roentgenographic examination of his pelvis and spine revealed rather advanced changes of atrophic arthritis in both hips but both sacro-iliac joints were practically normal. No change could be detected in the lumbar spine. We believe that this is really the first case of A. S. in which the sacro-iliac joints were roentgenographically normal; but we must point out that the spine itself was free from the typical changes of the disease.

III

Sacro-iliac Joints are Involved Earlier than the Spine.—To prove this point, we have extracted from the bulk of our clinical material, which amounts to 153 cases of A. S., 12 cases in which patients showing the clinical picture of this disease revealed some definite roentgenographic changes in their sacro-iliac joints *though the spine was free from any abnormality.* Let us sum up rapidly the principal features of these cases.

Age.—All our patients, but two, were observed around the age of 30 years and most of them had complained of functional symptoms in the spine since the age of from two to eight years. These findings confirm the view expressed by G. Scott that the disease belongs to adolescence in most cases.

Sex.—All our patients were males, which is an additional confirmation of the enormous preponderance of the disease among men.

mild tuberculosis of the lungs was noted; in five cases, no definite infection could be established in the past history.

Duration.—In most cases, the duration

Fig. 4. Dried specimen of a relatively early case of ankylosing spondylarthritis. The "syndesmophytes" (bridging) have not yet invaded the entire intervertebral spaces. Note bilateral sacro-iliac fusion.
(*Courtesy of Gazette Médicale de France.*)

Past History.—In spite of the fact that the existence of any focus of infection does not entail the certainty of its being the cause of any chronic disease occurring later on, we must point out that one-third of our patients had had some gonococcal infection, some time previously. In one case, was less than four years, but it is sometimes very difficult to make an accurate statement, since the early symptoms are slight and the progression of the disease very slow. It is impossible to state, even with some approximation, how long after the onset of the disease the first ossifica-

tions appear in the spine. In a young priest, aged 32 years, pains had appeared in the joints since the age of 12 years, with lumbar pains the past six years. It is remarkable that, as a rule, no pain was elicited by pressure at the site of the sacro-iliac joints.

Blood Changes.—In all cases without exception the sedimentation rate and the resorcine flocculation test were grossly abnormal and proved that an inflammatory condition was in process.

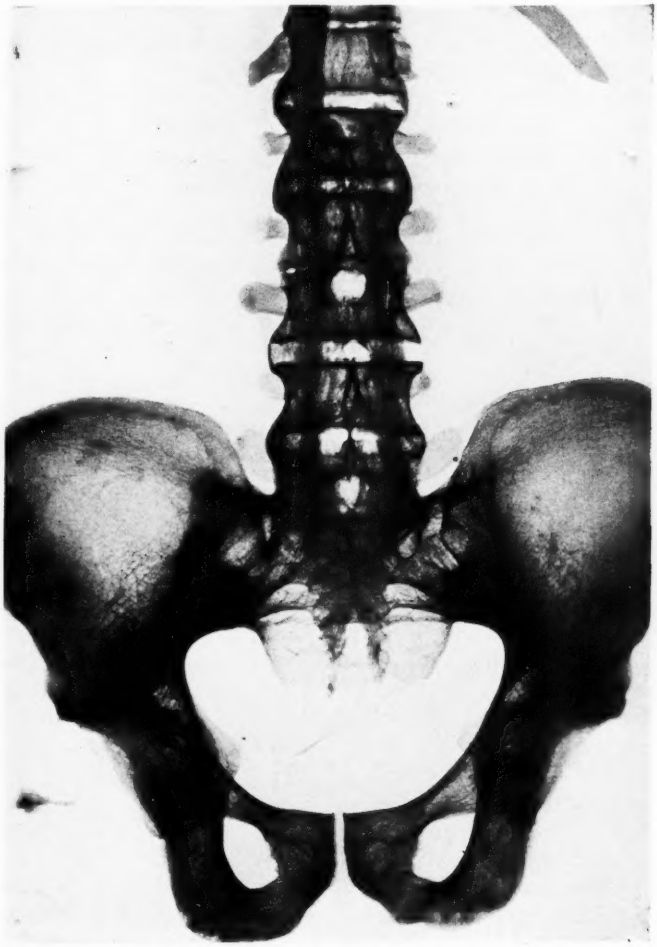

Fig. 5. Roentgenogram of same specimen taken in the same position as the photograph of Figure 4.

(*Courtesy of Gazette Médicale de France.*)

The spine was roentgenologically normal, though both sacro-iliacs were involved.

Clinical Picture.—In all our cases, the patients complained of pains and stiffness in the spine with muscular contracture and impairment in the chest expansion. By examination, tender areas were found along the spinous processes and on pressure at the site of some apophyseal (facets) joints.

IV

Roentgenographic Changes in the Sacro-iliac Joints in the Early Stages of Ankylosing Spondylarthritis.—The roentgeno-

graphic study of the sacro-iliac joints has been the subject of many papers during the past years. In almost every country, roentgenologists of experience have tried to find the technic best for the proper visualization of these joints. We shall not go into the details of the various technics since they are not of great value in the study of the early changes of the sacro-iliac joints in A. S. The reason is that most of these changes do not especially affect the joint space, but much more the adjacent bony structures of the sacrum and the ilium. These changes, as experience has shown us, are much more accurately detected on a routine frontal view of the pelvis (the sacrum lying against the film, the lumbar lordosis being reduced by bending the knees, the thighs in abduction). This technic shows the sacro-iliac joints' surfaces in their longest development as shown by Bărsony and Schülhoff (6). It affords a good comparison between both sides and, curiously enough, reveals more details in the structure of the bones adjoining the sacro-iliac joints than the special oblique views designed to map out the joint space of one sacro-iliac. This is true presumably because the abnormal area is projected on the film on a wider surface than when the roentgen rays strike the joint space tangentially.

Roentgenographic Changes.—These deal with the joint space, the contours of the bones, and the structure of the subchondral bone, these last two elements being of much greater importance than the first one. Indeed, the joint space is not, as a rule, thinned and does not become irregular by cartilage or bone erosion as in a true arthritis. We admit the difficulty of interpreting these early cases, since we know the great variability of the roentgenographic films of the sacro-iliac joints in normal persons. We want to emphasize that in some cases the changes which we describe do not, at the beginning, affect the entire length of the joint but only a part of it. The rest of the joint space appears to be absolutely normal. Later on, the entire joint becomes abnormal and the joint space disappears altogether very rapidly without passing through a stage of diminution of its width. To make our description more clear, we have classified our cases in three stages.

First Stage: Pseudo-widening of the Joint Space.—On a segment of one or both joints, generally in the lower segment, the contours of the subchondral bone become woolly and hazy; the clear-cut outline of the joint space on the iliac side disappears entirely. There is a loss of calcium alongside the margin of the bone. This *marginal decalcification* results in the appearance of a widening of the joint space (Fig. 1).

Second Stage: Picnotic Formation in the Joint Area.—The development of the first stage ends, after several months or a year, in a mottled appearance of the cancellous bone both on a wide area of the lateral portion of the sacrum and of the ilium. Some small spots appear very translucent, some look hypercalcified (in French: *aspect tigré*) (Fig. 2), or spotted.

Third Stage: Loss of Joint Space and Synostosis.—This is a far remote stage and a terminal one. In the previous stage the joint space is hardly visible on the mottled surface. At this stage, there is more uniformity in the calcification of the sacro-iliac region. Some thin ossified fibers appear transversely across the joint space and this fibrillary structure is often accompanied by osteosclerosis in the adjoining regions. In the course of the disease the bony densification tends to become more and more marked. This stage was rarely observed in our cases since it is generally a contemporary of the existence of the ossifications between the vertebræ. In the greatest majority of cases, such changes affect the whole length of both sacro-iliac joint spaces and are frequently accompanied by ossification of the iliolumbar ligaments.

Considering the changes in our 12 early cases, we find that in nine both sacro-iliacs were involved, but generally each of them partially. In the other three instances, the changes were unilateral and also partial.

V

Differential Diagnosis.—The association of clinical signs of lumbar pains and stiffness with roentgenographic changes in the sacro-iliacs, which do not give rise to any clinical symptoms, can hardly be mistaken for another pathologic condition.

What is commonly called *sacro-iliac arthritis* (*hypertrophic*), a clinical condition consisting of lumbar and pelvic pains appearing in elderly and stout women with weak muscles and ligaments, is a very peculiar syndrome which has hardly any point of conjunction with the subject we are considering. We almost never observe it in men, while these are affected with A. S. much more frequently than women. On examination of these patients, there is no real lumbar stiffness but only some muscular spasm, and the sacro-iliac joints are very tender under finger pressure; a sign which almost never occurs in A. S. The clinical symptoms, which are sometimes very violent, are in

Fig. 6. Same specimen as that shown in Figure 4. Right anterior oblique view. The vertebral ossifications are clearly seen frontally.
(*Courtesy of Gazette Médicale de France.*)

direct contrast with the absence of roentgenographic changes in the sacro-iliacs. Furthermore, we do not believe that this condition is a true arthritis but merely a joint insufficiency due to the fact that the static forces are by exception in only this joint of the body parallel to the joint space. It is easy to understand that any weakness in the sacro-iliac ligaments and the adjoining muscles will bring about a painful distention causing suffering.

Sacro-iliac tuberculous arthritis may occur in adults—in females as well as in males—and can be the cause of pains in the sacro-iliac region and of diverse nervous radiations in the lower limbs and the pelvis. A confusing point with A. S. is that this condition may be associated with an impairment of the general condition, low-grade temperature, and an accelerated sedimentation rate. In the early stages the roentgenographic findings are nega-

Fig. 7. Roentgenogram of same specimen taken in same position as the photograph of Figure 6. As the rays pass through the ossifications normally, the latter can scarcely be seen.

(*Courtesy of Gazette Médicale de France.*)

tive, but, later on, when they appear they consist of definite bone destruction in a limited area, an entirely different picture from any of the ones which we have described in A. S.

A question may arise as to whether or not there exists a congenital obliteration of one or both sacro-iliac joints. We have never observed it. It might be the result of a fetal infection and would be proved certainly much earlier in life than the time of onset of A. S.

Infectious sacro-iliac arthritis deserves a much more accurate consideration since this condition is very close to the one which we observe at the onset of A. S. The roentgenographic changes consist of a rather diffused area of decalcification, the joint space becoming less and less visible. But in the very few cases which have come under our observation, we have been struck by the constant association of clinical symptoms in the lumbar region associated with these findings, and at least two cases which were primarily labelled "sacro-iliac arthritis" a few years ago have proved, in the course of time, to be definite cases of A. S. In two additional cases the diagnosis of sacro-iliac arthritis had been laid down from four to six years previously by qualified physicians, and when they came under our observation they had developed unquestionable symptoms of A. S. It is well to mention that 12 cases reported as bilateral sacro-iliac obliteration by S. C. Woldenberg (7), 14 years ago, were certainly, for their greatest majority, early cases of A. S., since the clinical description of the cases with the preponderance of vertebral symptoms is absolutely typical of the disease. Roentgenographic changes of these cases described by Blaine, in 1923, are very similar to ours but no interpretation was given.

In 16 cases of A. S. detected by us, we were able to prove by re-interpreting the roentgenograms taken two, four, or even eight years previously, that sacro-iliac changes were already existent at that early stage but had been overlooked by qualified roentgenologists. Gilbert Scott has started a methodical survey in which he intends to radiograph all sacro-iliac joints of young men between the ages of 20 and 30 years who come to the Red Cross Clinic in London. It will be most interesting within a few years to find out, from among those who showed sacro-iliac changes of the type described by us, what percentage has developed A. S. We feel relatively sure that this percentage will be very high.

But we must remark that this disease has not such a chronic tendency as rheumatoid arthritis and may sometimes stop in its course even though untreated. We have seen a case in which, on the occasion of a roentgenographic examination, one sacro-iliac joint was obliterated and two ossifications were detectable between the third, fourth, and fifth lumbar vertebræ. Ten years previously, this man, about 25 years of age, had undergone a period of chronic lumbago for two years. Without any treatment the disease had been arrested before giving rise to its essential clinical symptoms on the spine.

VI.—DISCUSSION

After reviewing 153 cases of A. S., some of which we have followed during many years, we have been struck by the following features of the disease:

(*A*) It occurs very rarely in women.

(*B*) Patients frequently give a previous history of genital infection: prostate, seminal vesicles, or rectosigmoidal disturbances.

(*C*) The disease has a very low-grade evolution, the onset often being difficult to determine. It progresses upward from the pelvis to the cervical region, and to the joints of the roots of the limbs.

(*D*) The roentgenographic changes in the sacro-iliacs are contemporary of the early and confused low back symptoms and precede, in most cases, the vertebral involvement.

From these deductions, we have tried, since four years ago (8), to present a pathogenesis of the disease which would explain completely its special features.

We believe that the primary focus in A. S. is in the genito-urinary system or in the low part of the bowels, and that the toxic products excreted by this focus are drained into the lymphatic system of the pelvis and from this place alongside the spine. In the male, the lymphatic vessels from the prostate and the seminal vesicles pass, on both sides of the medial line, in front of each sacro-iliac joint, and extend upward in the posterior part of the abdomen on both sides of the spinal column just in front of the apophyseal joints with which they have many connections. They are directly applied on the vertebral column behind the aorta and the vena cava.

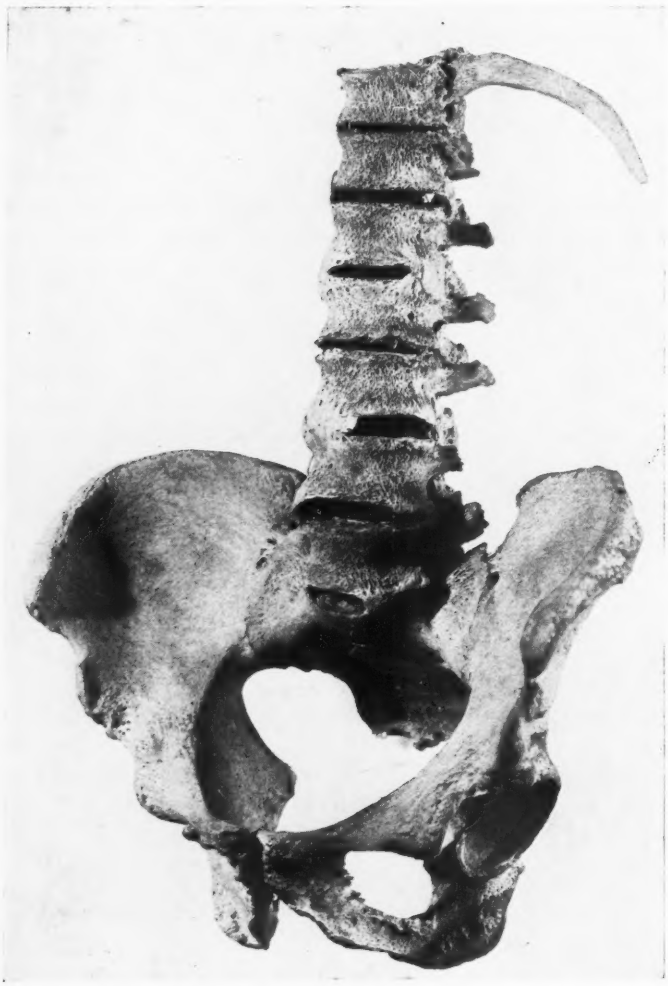

Fig. 8. Same specimen as that shown in Figure 4. Left anterior oblique view. The vertebral ossifications can hardly be seen.
(*Courtesy of Gazette Médicale de France.*)

In the female, the lymphatic vessels of the uterus and the vagina follow approximately the same path and also lie near the intervertebral joints. Conversely, the lymphatic vessels of the Fallopian tubes and of the ovaries are much more laterally

situated in the pelvis, and when they come up into the abdomen they lie on the ventral aspect of the large blood vessels—aorta and vena cava. They have very few anastomotic connections with the rest of the females. This theory explains quite satisfactorily the slow progression of the disease; first to the sacro-iliac joints and later ascending along the different segments of the spine.

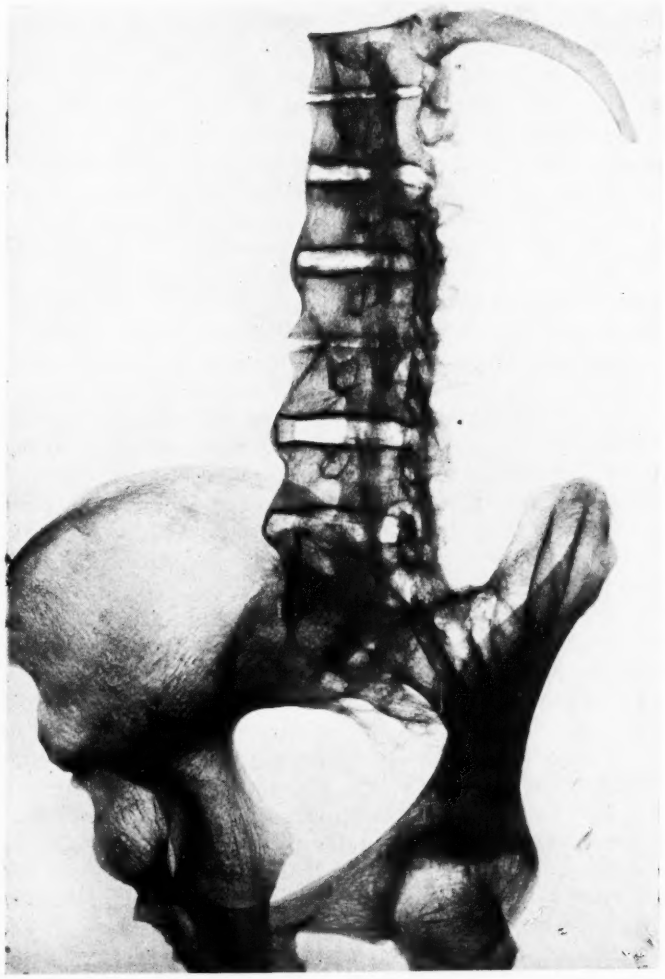

Fig. 9. Roentgenogram of same specimen taken in same position as in Figure 8. As the roentgen rays fall upon the ossifications tangentially, the latter are best seen. Compare with Figure 7.
(*Courtesy of Gazette Médicale de France.*)

retro-aortic system (Rouvières). Since we know that the draining of the uterus and vagina is far easier than that of the ovaries and tubes, we can understand why the occurrence of A. S. is so infrequent among

VII.—BRIEF COMMENTS ON INTERVERTEBRAL OSSIFICATIONS

Every physician is aware of the typical bamboo-spine which is the roentgenographic feature of A. S. in its terminal

stage. But there are still many mistakes made in the earlier stages when only a few ossifications have appeared. Most of them are described as osteophytes and no special attention is given them. Thus, the serious condition of a total ankylosis of the spine which may appear later is thoroughly overlooked. We have given the name of "syndesmophytes" (9) to those ossifications which, from the very beginning of their onset, have a tendency to unite two adjacent vertebræ (bridging). Syndesmophytes can be easily distinguished from the osteophytes from the beginning since the former appear as a woolly shadow in the intervertebral space which, within a few months, is transformed into a rather clear-cut, dense, linear calcification without a cortex having the appearance of a thin or a thick comma, added to the contour of the vertebral body. The osteophytes on the contrary have a much thicker base, are covered with a cortex which comes from that of the vertebral body, and a structure of cancellous bone like the vertebral body itself.[3] Since the opportunity has been rarely given of checking the roentgenographic findings with the pathologic specimens of A. S., the difference in these structures in their early stages could not be proved. We had the good fortune of viewing a spondylarthritic spine of rare specimen in which the ossifications had not yet completely invaded all the intervertebral spaces (10). The photographs and the roentgenograms of this specimen at various angles are reproduced herein (Figs. 4 to 9). It will be easy, from this comparative study to understand why our diagnostic findings in A. S. are relatively limited. Most of the thin layers of dense bone uniting the vertebræ at the periphery are not shown in the roentgenograms when these are in the normal path of the roentgen ray. It is only when a tangential ray strikes one of these pathologic structures that its opacity is sufficient to appear on the film, and one little opaque comma generally indicates a very wide ossified structure. These figures show also that for the certain detection of these vertebral ossifications at an early stage, several films must be taken at different angles—frontal, lateral, oblique.

[3] This term means a growth from the ligament, in opposition to "osteophyte" which means a growth from the bone.

VIII.—THERAPEUTIC DEDUCTIONS

The preceding considerations as to the early diagnosis of A. S. are not merely academic. Every chronic disease must be treated at the beginning and not at the end, and experience has proved that A. S., when actively treated in its pre-ankylosing period, is a curable condition.

Among the treatment methods proposed for this disease, we must recall briefly the usual measures, especially physiotherapy, which, for many physicians, is the basis of treatment. The most important measure is functional rest which is obtained by prolonged decubitus; it may be improved by the use of a plaster shell supporting the whole spine, a method applied by the Boston Orthopedic School and emphasized by Loring Swaim (3). Among the other therapeutic measures, the use of heat to the spine by electric lamps, short waves, or diathermy certainly has an analgesic value. At Aix-les-Bains, in the active periods, we use reclining baths or deep baths associated with undercurrent spray (*douche sous-marine*). When the disease has partially subsided, hot showers (*bouillon*) associated with gentle douche-massage are of definite value to improve the mobility of the spine and counteract muscular atrophy.

But in our experience, two internal treatments have proved of very great value, not only as symptomatic measures, but for the treatments of the disease itself. They are gold salts injections (chrysotherapy) and radio-active injections (thorium X or radon).

Chrysotherapy.—Since we introduced gold salts in the treatment of rheumatoid arthritis more than ten years ago, this method has spread in almost every country and is being used to date in the United States. A. S., though a different disease from rheumatoid, belongs to the group of

atrophic and inflammatory arthritis and thus is amenable to gold salts treatment. We use it especially in active stages of the disease when the clinical symptoms of pain and stiffness are associated with a high sedimentation rate. I described the technic of these injections four years ago in the *Journal of Laboratory and Clinical Medicine* (11). Intravenous injections are not necessary and we generally use intramuscular injections of myochrysine, allochrysine, myoral, or solganal B. Since the action of these different compounds is very similar, moderate doses not exceeding 10 cg. per week and 1 gr. 20 for one series, are sufficient. But the series must be repeated at two- or three-month intervals as long as the disease remains active.

Radio-active Injection.—The use of radio-active preparations injected intramuscularly or intravenously was introduced in the treatment of A. S. by means of thorium by A. Léri, 14 years ago. These injections are given at weekly intervals with a rising dosage from 50 to 150 or 250 micrograms. A series consisting of eight or ten injections and totalling between 800 and 1,200 micrograms are given. As thorium X does not seem to be available in many countries, we have in the past three years used gaseous injections of radon. The latter is produced by small metallic tubes containing a definite amount of radium element. When the proper amount of radon has accumulated in the closed tube, it is connected on one side with a hypodermic needle, and on the other side with a 20 c.c. syringe of oxygen or even sterilized air. By pushing the latter through the tube the radon is expelled, injected under the skin, and promptly absorbed by the general circulation. We have used a dosage of from 6,000 to 12,000 millimicrocuries, given every second day, with a total of from 12 to 20 injections for one series. The series may be repeated after an interval of from four to six months. We have observed no untoward effect.

The use of radon is reserved to the less active stages of the disease and also to cases of longer standing.

The results of the internal treatment of A. S. in its early stages by the use of gold salts or radon or by alternate series of each have been most satisfactory. After a few weeks or a month or two, the vertebral pains subside progressively and the motion of the spine increases. This is not a purely symptomatic action; if the treatment is pursued for one or two years, by repeated series, the arrest of the disease may be total. Among 25 cases treated by this method in the pre-ankylosing period which have remained under observation during two to five years, 80 per cent have been made thoroughly free of symptoms after one or two years' treatment. Only 15 per cent have experienced some relapse, which could be checked by resuming treatment. In only a few cases, some vertebral ossifications (syndesmophytes) have appeared after the beginning of the treatment. Most of the patients have retained a certain amount of vertebral stiffness but the absence of any pain has permitted an almost complete functional activity.

We feel that if every case of A. S. were treated in its early stage along these lines, with patience and perseverance, the picture of *spondylose rhizomélique*, with a total ankylosis of the whole spine and the sacro-iliac hip and shoulder joints would become an extreme rarity.

SUMMARY

1. From the examination of 153 cases of ankylosing spondylarthritis, it appears that sacro-iliac joints are roentgenographically involved in over 98 per cent of the cases.

2. From the examination of 12 cases observed in the very early stages of the disease, evidence is given that sacro-iliac joints are roentgenographically involved previously to any changes of the spine. The reverse has not been observed.

3. A description of the roentgenographic changes in the sacro-iliac joints is given.

4. An hypothesis is presented which explains the causes, the onset, and the progress of ankylosing spondylarthritis.

5. A roentgenographic differentiation is given between the non-ankylosing vertebral ossifications (osteophytes) and the ankylosing ossifications (syndesmophytes).

6. The practical value of these data is justified by the good results obtained in the treatment of the early stages of ankylosing spondylarthritis, *i.e.* (*a*) by gold salts injections, and (*b*) by radio-active injections (thorium X and radon).

REFERENCES

(1) FORESTIER, J.: Le Diagnostic des Débuts de la Spondylose Rhizomélique. Concours Méd., **13,** March, 29, 1936.

(2) KREBS, W.: Klinik der sogenannten rheumatischen Wirbelsaulenerkrankungen. Rheuma probleme. Band II, p. 3, Georg Thieme, Leipzig, 1931.

(3) SWAIM, L. T., and KUHNS, J. G.: Treatment of Chronic Arthritis of the Spine. Arch. Phys. Ther., **13,** 517–527, September, 1932.

(4) SCOTT, S. G.: Chronic Infection of the Sacro-iliac Joints as a Possible Cause of Spondylitis Adolescens. British Jour. Radiol., **9,** 126–131, February, 1936.

(5) BUCKLEY, C. W.: Ankylosing Spondylarthritis. Rep. on Chronic Rheumatic Dis., **1,** 87–89, 1935.

(6) BÄRSONY, T., and SCHÜLHOFF, O.: Wie liegt die Sakroiliakgelenkeim Röntgenbilde. Röntgenpraxis, **7,** 1935.

(7) WOLDENBERG, S. C.: Bilateral Sacro-iliac Obliteration. Jour. Am. Med. Assn., **82,** 1027, 1028, March 29, 1924.

(8) FORESTIER, J.: Nature de la Spondylose Rhizomélique. Rev. du Rhumatism, June, 1935.

(9) FORESTIER, J., and ROBERT, P.: Ostéophytes et Syndesmophytes. Gaz. Méd. de France (supp. Radiol.), pp. 192–202, May 1, 1934.

(10) FORESTIER, J., COLIEZ, R., and ROBERT, P.: Etudes radiologiques de Pièces Sèches de Rhumatisme Vertébral. Gaz. Méd. de France (supp. Radiol.), **43,** 139–145, Oct. 1, 1936.

(11) FORESTIER, J.: Rheumatoid Arthritis and its Treatment by Gold Salts: Results of Six Years' Experience. Jour. Lab. and Clin. Med., **20,** 827–840, May, 1935.

RADIOLOGICAL SERVICES FOR CRIPPLED CHILDREN

Stimulated by free grants-in-aid from federal funds appropriated under Title 5, part 2, of the Social Security Act, 47 States and Alaska, Hawaii, and the District of Columbia have enacted tax measures for the rehabilitation of crippled children. When approved by the Children's Bureau of the U. S. Department of Labor, the state plans thus created receive a grant from the Federal Government based upon the need in each State but in no case exceeding the amount raised and available for expenditure within the State.

Practically every crippled child requires a roentgen examination before remedial measures may be begun. The operation of these plans is, therefore, of very great importance to radiologists and considerable attention has been given to the methods through which radiological services are provided to beneficiaries of the act.

Recently the Inter-Society Committee for Radiology made a study of representative plans in a number of States to determine the status of radiology in the operation of these plans and the manner in which radiologists are compensated for their service. No two plans are exactly alike and a wide variety of methods are employed for the administration of the plans and the remuneration of orthopedists, pediatricians, plastic surgeons, radiologists, and others who may be called upon to render service authorized under the State acts and approved by the Federal department.

In a considerable number of States, orthopedists alone are remunerated on a fee schedule or a part-time basis for services to crippled children. In most such plans, provision is made for the payment of a consultant's fee of from five to ten dollars when another specialist is called in to assist in the diagnosis or treatment of the patient's condition. In others, provision is made for payment to pediatricians and plastic surgeons, while in about half of the plans in operation, provision is made for payment to radiologists on a fee-schedule basis. Whenever a fee schedule has been adopted, it will usually be found to be on a reduced basis, in some cases amounting to 50 per cent of the regular fee for private patients or an amount about equal to the cost of rendering the service.

Benefits of the crippled children's program are confined, presumably, to children whose parents are unable to pay the cost for private treatment. In several States, children are admitted for diagnosis of their condition before their economic eligibility is considered. In these States, the authorities declare that the ability of the parent to pay for treatment cannot be accurately determined until diagnosis is made and the probable cost of such treatment can be known. In these States, therefore, the services of the roentgenologist is called upon before any consideration is given to the patient's ability to pay for private treatment.

In the great majority of States, the plan is administered by the Department of Health. In a number of States, the Vocational Department or the Department of Public Welfare has the responsibility for administration of the plan. In one State, Iowa, the program is carried on entirely under the administration of the State University.

From a questionnaire sent to 48 state representatives of the Inter-Society Committee, 24 replies were received answering specific questions concerning the operation of the crippled children's program and the methods for securing radiological diagnosis. In seven States no provision is made for the remuneration of radiologists rendering service to beneficiaries of the act. Some of these apparently do not contemplate the necessity for radiological examinations while others apparently presume it to be a part of hospitalization. In Arkansas, Georgia, Kansas, North Carolina, Oklahoma, Tennessee, and Virginia it is reported that no provision is made for radiological services. In these States, therefore, the administrators of the plan regard radiology as a part of the hospital accommodations and expect roentgen diagnosis to be included by the hospital for the *per diem* payment it receives for hospitalized children.

One correspondent reports that "We are informed that the federal agency takes the

position that the hospitals pay the roentgenologist stipulated salaries; hence, no individual payments are made." The information this correspondent has received is obviously inaccurate, in view of the fact that a number of States do provide for the payment of fees for radiological services under plans that are approved by the Children's Bureau. The decision reached in these States is, therefore, a local and arbitrary one and has not been influenced by the federal authorities charged with the approval and advisory control of state plans.

In Alabama, California, Colorado, Connecticut, Indiana, Kentucky, Michigan, Minnesota, Montana, Nebraska, Oregon, Texas, Utah, Vermont, and Wisconsin, correspondents reported that the state administrative agency provides for payment for radiological services performed on crippled children. In most of these, payments are made according to an adopted fee schedule. As stated above, these fee schedules are customarily on a reduced basis and the maximum amount allowed for any single examination is usually around ten dollars. In a few States, it is no higher than five dollars per examination. In some States, a complete schedule has been approved, setting forth the fee allowed for various procedures. In a few States, the fee schedule is used only for out-patients or for after care and check-ups. Radiological services in these plans are included in the *per diem* payment allowed to hospitals while the beneficiary is confined to the hospital.

Included in the regulations of nearly every state plan is the provision that specialists performing services for the beneficiaries of the act must be certified by their national examining board and in such cases radiologists are not permitted to receive remuneration for their services unless they are diplomates of the American Board of Radiology.

In North Carolina, it is reported that no arrangement is made for payment to radiologists while anesthetists are remunerated for services rendered on a fee-schedule basis. The same is no doubt true in some other plans in which no provision is made for radiological fees.

To those radiologists in States where no provision is made for fees for radiological services, the Inter-Society Committee suggests that conferences be held with the local authorities in an effort to secure a more equitable arrangement. All these projects are admittedly of a charitable nature and fees should be reduced to a minimum, but there is no good reason why recognition should not be given to the important rôle of roentgen diagnosis as a necessary preliminary to the institution of orthopedic measures. For services rendered to both in-patients and out-patients, arrangements should be made for paying for radiological diagnosis on a reduced fee schedule. Even in those institutions where radiological fees for services rendered to hospitalized patients is customarily collected by the hospital, it is unfair to both the hospital and the radiologist to expect these services to be rendered as a part of hospital care and included in the *per diem* allotment in the case of beneficiaries of the crippled children's program.

MAC F. CAHAL,
Executive Secretary

RADIOLOGICAL SOCIETIES IN THE UNITED STATES

Editor's Note.—Will secretaries of societies please cooperate with the Editor by supplying him with information for this section? Please send such information to Leon J. Menville, M.D., 1201 Maison Blanche Bldg., New Orleans, La.

CALIFORNIA

California Medical Association, Section on Radiology.—Chairman, Karl M. Bonoff, M.D., 1930 Wilshire Blvd., Los Angeles; *Secretary,* Carl D. Benninghoven, M.D., 95 S. El Camino Real, San Mateo.

Los Angeles County Medical Association, Radiological Section.—President, E. N. Liljedahl, M.D., 1322 North Vermont Ave., Los Angeles; *Vice-president,* M. L. Pindell, M.D., 678 South Ferris Ave.; *Secretary,* Wilbur Bailey, M.D., 2007 Wilshire Blvd.; *Treasurer,* Henry Snure, M.D., 1414 South Hope Street. Meets every second Wednesday of each month at County Society Building.

Pacific Roentgen Club.—Chairman, Karl M. Bonoff, M.D., Los Angeles; *Members of Executive Committee,* I. S. Ingber, M.D., A. C. Siefert, M.D., D. R. MacColl, M.D.; *Secretary-Treasurer,* L. Henry Garland, M.D., 450 Sutter St., San Francisco. Executive Committee meets quarterly; Club meets annually during annual session of the California Medical Association.

San Francisco Radiological Society.—Secretary, L. H. Garland, M.D., 450 Sutter Street. Meets monthly on first Monday at 7:45 P.M., alternately at Toland Hall and Lane Hall.

COLORADO

Denver Radiological Club.—President, F. B. Stephenson, M.D., 452 Metropolitan Bldg.; *Vice-president,* K. D. A. Allen, M.D., 452 Metropolitan Bldg.; *Secretary,* E. A. Schmidt, M.D., 4200 E. Ninth Ave.; *Treasurer,* H. P. Brandenburg, M.D., 155 Metropolitan Bldg. Meets third Tuesday of each month at homes of members.

CONNECTICUT

Connecticut State Medical Society, Section on Radiology.—Chairman, Samuel M. Atkins, M.D., 63 Central Ave., Waterbury; *Secretary-Treasurer,* Max Climan, M.D., 242 Trumbull St., Hartford. Meetings twice annually in May and September.

DELAWARE

Affiliated with Philadelphia Roentgen Ray Society.

FLORIDA

Florida Radiological Society.—President, H. B. McEuen, M.D., Jacksonville; *Vice-president,* Joseph H. Lucinian, M.D., Miami; *Secretary-Treasurer,* John N. Moore, M.D., 210 Professional Bldg., Ocala. Meetings held in November and at the annual meeting of the Medical Association of Florida in the spring.

GEORGIA

Georgia Radiological Society.—President, James J. Clark, M.D., Doctors Bldg., Atlanta; *Vice-president,* L. P. Holmes, M.D., University Hospital, Augusta; *Secretary-Treasurer,* Robert C. Pendergrass, M.D., Prather Clinic, Americus. Meetings twice annually, in November and at the annual meeting of the Medical Association of Georgia in the spring.

ILLINOIS

Chicago Roentgen Society.—President, Roe J. Maier, M.D.; *Vice-president,* Adolph Hartung, M.D.; *Secretary,* Chester J. Challenger, M.D., 3117 Logan Blvd. Meetings the second Thursday of each month from October to May, except December, at the Hotel Sherman.

Illinois Radiological Society.—President, Cesare Gianturco, M.D., 602 W. University Ave., Urbana; *Vice-president,* Fred H. Decker, M.D., 802 Peoria Life Bldg., Peoria; *Secretary-Treasurer,* Edmund P. Halley, M.D., 968 Citizens Bldg., Decatur. Meetings quarterly by announcement.

Illinois State Medical Society, Section on Radiology.—The next meeting will be in Peoria, in May, 1940. The officers are: *Chairman,* Warren W. Furey, M.D., 6844 Oglesby Ave., Chicago; *Secretary,* Harry W. Ackemann, M.D., 321 W. State St., Rockford.

INDIANA

The Indiana Roentgen Society.—President, Juan Rodriguez, M.D., 2902 Fairfield Ave., Fort Wayne; *President-elect,* H. H. Inlow, M.D., Shelbyville; *Vice-president,* Wemple Dodds, M.D., Crawfordsville; *Secretary-Treasurer,* Clifford C. Taylor, M.D., 23 E. Ohio St., Indianapolis. Annual meeting in May.

IOWA

The Iowa X-ray Club.—Holds luncheon and business meeting during annual session of Iowa State Medical Society.

KENTUCKY

Kentucky Radiological Society.—President, D. B. Harding, M.D., Lexington; *Vice-president,* I. T. Fugate, M.D., Louisville; *Secretary-Treasurer,* Joseph C. Bell, M.D., 402 Heyburn Bldg., Louisville. Meeting annually in Louisville, third Sunday afternoon in April.

MAINE

See New England Roentgen Ray Society.

MARYLAND

Baltimore City Medical Society, Radiological Section.—Chairman, Whitmer B. Firor, M.D., 1100 N. Charles St.; *Secretary,* Walter L. Kilby, M.D., 101 W. Read St. Meetings third Tuesday of each month.

MASSACHUSETTS

See New England Roentgen Ray Society.

MICHIGAN

Detroit X-ray and Radium Society.—President, Sam W. Donaldson, M.D., 326 N. Ingalls St., Ann Arbor;

Vice-president, Clarence Hufford, M.D., 421 Michigan Ave., Toledo, Ohio; *Secretary-Treasurer*, E. R. Witwer, M.D., Harper Hospital, Detroit. Meetings first Thursday of each month from October to May, inclusive, at Wayne County Medical Society club rooms, 4421 Woodward Ave.

Michigan Association of Roentgenologists.—President, C. K. Hasley, M.D., 1429 David Whitney Bldg., Detroit; *Vice-president*, M. R. Cooley, M.D., Mercy Hospital, Jackson; *Secretary-Treasurer*, C. S. Davenport, M.D., 609 Carey St., Lansing. Meetings quarterly by announcement.

MINNESOTA

Minnesota Radiological Society.—President, Leo G. Rigler, M.D., University Hospital, Minneapolis; *Vice-president*, Harry M. Weber, M.D., Mayo Clinic, Rochester; *Secretary*, John P. Medelman, M.D., 572 Lowry Medical Arts Bldg., St. Paul. These officers will assume their duties after the Summer meeting which will be held in connection with the Minnesota State Medical Society, May 31 to June 2, 1939.

MISSOURI

The Kansas City Radiological Society.—President, L. G. Allen, M.D., 907 N. 7th St., Kansas City, Kansas; *Secretary*, Ira H. Lockwood, M.D., 306 E. 12th St., Kansas City, Mo. Meetings last Thursday of each month.

The St. Louis Society of Radiologists.—President, Paul C. Schnoebelen, M.D.; *Secretary*, W. K. Mueller, M.D., University Club Bldg. Meets on fourth Wednesday of October, January, March, and May, at a place designated by the president.

NEBRASKA

Nebraska Radiological Society.—President, T. T. Harris, M.D., Clarkson Memorial Hospital, Omaha; *Secretary*, D. Arnold Dowell, M.D., 117 S. 17th St., Omaha. Meetings first Wednesday of each month at 6 P.M. in Omaha or Lincoln.

NEW ENGLAND ROENTGEN RAY SOCIETY

(Maine, New Hampshire, Vermont, Massachusetts, and Rhode Island.) *President*, Langdon T. Thaxter, M.D., Maine General Hospital, Portland, Maine; *Secretary*, Aubrey O. Hampton, M.D., Massachusetts General Hospital, Boston. Meetings third Friday of each month from October to May, inclusive, usually at Boston Medical Library.

NEW HAMPSHIRE

See New England Roentgen Ray Society.

NEW JERSEY

Radiological Society of New Jersey.—President, P. S. Avery, M.D., Middlesex Hospital, New Brunswick; *Vice-president*, J. G. Boyes, M.D., 912 Prospect Ave., Plainfield; *Treasurer*, H. A. Vogel, M.D., 1060 E. Jersey St., Elizabeth; *Secretary*, W. James Marquis, M.D., 198 Clinton Ave., Newark; *Counsellor*, A. W. Pigott, M.D., Skillman. Meetings at Atlantic City at time of State Medical Society, and Midwinter in Newark as called by president.

NEW YORK

Associated Radiologists of New York, Inc.—President, Henry A. Barrett, M.D., 140 East 54th St., New York City; *President-elect*, I. J. Landsman, M.D., 910 Grand Concourse, New York City; *Vice-president*, Frederic E. Elliott, M.D., 122 76th St., Brooklyn; *Treasurer*, Solomon Fineman, M.D., 133 East 58th St., New York City; *Secretary*, William J. Francis, M.D., 210 Fifth Ave., New York City. Regular meetings the first Monday evening of the month in March, May, October, and December.

Brooklyn Roentgen Ray Society.—President, A. L. L. Bell, M.D., Long Island College Hospital, Henry, Pacific, and Amity Sts.; *Secretary-Treasurer*, L. J. Taormina, M.D., 1093 Gates Ave. Meetings first Tuesday in each month at place designated by president.

Buffalo Radiological Society.—President, Chester D. Moses, M.D., 333 Linwood Ave.; *Vice-president*, Edward C. Koenig, M.D., 100 High St.; *Secretary-Treasurer*, Joseph S. Gian-Franceschi, M.D., 610 Niagara St. Meetings second Monday evening each month, October to May, inclusive.

Central New York Roentgen Ray Society.—President, Jesse Randolph Pawling, M.D., 305 Clinton St., Watertown; *Vice-president*, Albert Lenz, M.D., 613 State St., Schenectady; *Secretary-Treasurer*, Carlton F. Potter, M.D., 425 Waverly Ave., Syracuse. Meetings are held in January, May, and October, as called by Executive Committee.

Long Island Radiological Society.—President, Samuel G. Schenck, M.D., Brooklyn; *Vice-president*, G. Henry Koiransky, M.D., Long Island City; *Secretary*, Marcus Wiener, M.D., 1430 48th St., Brooklyn; *Treasurer*, Louis Goldfarb, M.D., 608 Ocean Ave., Brooklyn. Meetings fourth Thursday evening each month at Kings County Medical Bldg.

New York Roentgen Society.—President, Harry M. Imboden, M.D., 30 W. 59th St., New York City; *Vice-president*, Henry K. Taylor, M.D., 667 Madison Ave., New York City; *Secretary*, Roy D. Duckworth, M.D., 170 Maple Ave., White Plains, N. Y.; *Treasurer*, Eric J. Ryan, M.D., St. Luke's Hospital, New York City.

Rochester Roentgen-ray Society.—Chairman, Joseph H. Green, M.D., 277 Alexander St.; *Secretary*, S. C. Davidson, M.D., 277 Alexander St. Meetings at convenience of committee.

NORTH CAROLINA

Radiological Society of North Carolina.—President, Robert P. Noble, M.D., 127 W. Hargett St., Raleigh; *Vice-president*, A. L. Daughtridge, M.D., 144 Coast

Line St., Rocky Mount; *Secretary-Treasurer*, Major I. Fleming, M.D., 404 Falls Road, Rocky Mount. Meetings with State meeting in May, and meeting in October.

OHIO

Cleveland Radiological Society.—President, J. H. West, M.D., 10515 Carnegie Ave.; *Vice-president*, Harry Hauser, M.D., City Hospital; *Secretary-Treasurer*, H. A. Mahrer, M.D., 10515 Carnegie Ave. Meetings at 6:30 P.M. at the Mid-day Club, in the Union Commerce Bldg., on fourth Monday of each month from October to April, inclusive.

Radiological Society of the Academy of Medicine (Cincinnati Roentgenologists).—*President*, B. M. Warne, M.D., Doctors Building, Cincinnati; *Secretary-Treasurer*, Justin E. McCarthy, M.D., 707 Race St., Cincinnati, Ohio. Meetings held third Tuesday of each month.

PENNSYLVANIA

Pennsylvania Radiological Society.—President, Louis A. Milkman, M.D., Medical Arts Bldg., Scranton; *First Vice-president*, James E. Ginter, M.D., Dubois; *Second Vice-president*, Alexander Stewart, M.D., Shippensburg; *Secretary-Treasurer*, L. E. Wurster, M.D., 416 Pine St., Williamsport; *President-elect*, Harvey N. Mawhinney, M.D., 6546 Darlington Road, Pittsburgh; *Editor*, William E. Reiley, M.D., Clearfield; *Assistant Editor*, Sydney J. Hawley, M.D., Danville.

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The Pittsburgh Roentgen Society.—President, Zoe A. Johnston, M.D., 601 Jenkins Arcade; *Vice-president*, Prentiss A. Brown, M.D., and *Secretary-Treasurer*, Harold W. Jacox, M.D., 4800 Friendship Ave. Meetings held second Wednesday of each month at 4:30 P.M., from October to June at various hospitals designated by program committee.

RHODE ISLAND

See New England Roentgen Ray Society.

SOUTH CAROLINA

South Carolina X-ray Society.—President, Percy D. Hay, Jr., M.D., McLeod Infirmary, Florence; *Secretary-Treasurer*, Hillyer Rudisill, Jr., M.D., Roper Hospital, Charleston. Meetings in Charleston on first Thursday in November, also at time and place of South Carolina State Medical Association.

SOUTH DAKOTA

Meets with Minnesota Radiological Society.

TENNESSEE

Memphis Roentgen Club.—Chairmanship rotates monthly in alphabetical order. Meetings second Tuesday of each month at University Center.

Tennessee Radiological Society.—President, Steve W. Coley, M.D., Methodist Hospital, Memphis; *Vice-president*, Eugene Abercrombie, M.D., 305 Medical Arts Bldg., Knoxville; *Secretary-Treasurer*, Franklin B. Bogart, M.D., 311 Medical Bldg., Chattanooga. Meeting annually with State Medical Society in April.

TEXAS

Texas Radiological Society.—President, Jerome H. Smith, M.D., San Antonio; *President-elect*, C. F. Crain, M.D., Corpus Christi; *First Vice-president*, M. H. Glover, M.D., Wichita Falls; *Second Vice-president*, G. D. Carlson, M.D., Dallas; *Secretary-Treasurer*, Henry C. Harrell, M.D., 517 Pine St., Texarkana. Meets annually. Temple is place of next meeting, Oct. 20 and 21, 1939.

VERMONT

See New England Roentgen Ray Society.

VIRGINIA

Radiological Society of Virginia.—President, Fred M. Hodges, M.D., 100 W. Franklin St., Richmond; *Vice-president*, L. F. Magruder, M.D., Raleigh and College Aves., Norfolk; *Secretary*, V. W. Archer, M.D., University of Virginia Hospital, Charlottesville.

WASHINGTON

Washington State Radiological Society.—President, H. E. Nichols, M.D., Stimson Bldg., Seattle; *Secretary*, T. T. Dawson, M.D., Fourth and Pike Bldg., Seattle. Meetings fourth Monday of each month at College Club.

WISCONSIN

Milwaukee Roentgen Ray Society.—President, H. W. Hefke, M.D.; *Vice-president*, Frederick C. Christensen, M.D.; *Secretary-Treasurer*, Irving I. Cowan, M.D., Mount Sinai Hospital, Milwaukee. Meets monthly on first Friday at the University Club.

Radiological Section of the Wisconsin State Medical Society.—Secretary, Russel F. Wilson, M.D., Beloit Municipal Hospital, Beloit. Two-day annual meeting in May and one day in connection with annual meeting of State Medical Society, in September.

University of Wisconsin Radiological Conference.—Secretary, E. A. Pohle, M.D., 1300 University Ave., Madison, Wis. Meets every Thursday from 4 to 5 P.M., Room 301, Service Memorial Institute.

EDITORIAL

LEON J. MENVILLE, M.D., *Editor* HOWARD P. DOUB, M.D., *Associate Editor*

SOME PRESENT AND FUTURE PROBLEMS OF RADIOLOGY[1]

In contemplating the present status of radiology one is naturally led to consider the enviable position which this relatively youthful member of the medical profession occupies. It is just 43 years since Roentgen first saw the fluorescence of the crystals which his trained eyes correctly interpreted as the manifestation of hitherto unrecognized rays. That event transcends in importance any discovery made in our lifetime. It extended the spectrum of electromagnetic radiation to hitherto unbelievably shorter wave lengths. The ionization of air by x-rays led to the recognition of the electron. From the electron has come the radio and a host of mechanical devices that depend upon the electronic principle. This is in addition to the purely medical uses of x-rays, upon which the life and health of people depend to such a large degree.

Emerson has said that "An institution is the lengthened shadow of one man." It is readily apparent that the recognition and the physical characteristics of x-rays are the lengthened shadow of one man—Roentgen. However, the shadows cast by the roentgen rays of to-day were neither entirely foreseen nor brought to full fruition by that great scientist alone. A host of scientists and physicians have indelibly imprinted their shadows along the path of radiation until it stretches into fields undreamed of by its discoverer. Time is not available to discuss the part played by these individual men, but the epochal discoveries resulting from their labors have made radiology the great specialty that it is to-day. These discoveries were not made without the exaction of a terrific toll of suffering and death of many of these pioneers. This specialty is so new that most of us have known intimately one or more of these men who have suffered so silently the pain and anguish attendant on this condition.

Ours is a great heritage which has come down to us as a result of the labors and sufferings of a host of brilliant pioneers. These men have builded on the solid foundation of scientific facts By a combination of perseverance and ingenuity they have unearthed many of the hidden secrets of disease processes and made them visually perceptible. At the same time, these mysterious rays were applied as a curative agent in various disease processes until to-day it is difficult to determine in which field their greatest successes lie. We have here, then, a great bilateral diagnostic and curative agent, the like of which the world has never seen.

We owe a great debt to the pioneer workers in radiology and only by conscientiously attacking the present and future problems that hinder the progress of this work can we uphold the high standards which they set, and partly repay this debt. It behooves us, then, to attempt a careful analysis of the situation in which radiology finds itself at present and to plan by every means at our disposal to consolidate our present position and to adopt measures calculated to advance our position to the front ranks of the medical profession. What, then, shall we do to accomplish this aim?

First, we must induce the best of our interne groups to enter radiology for training. As the finished product so largely depends upon the raw material which goes into it, so must the future of radiology depend upon the young men who will enter its ranks for training. It is lamentable that radiology is not attracting a larger percentage of the outstanding young men from the student and interne groups to its fold. This is attributable to a number of factors, the chief of which is a feeling of the lack of economic security and the fear of domination by outside groups. In many instances it is apparent to these younger men that the position of the radiologist compares unfavorably with that of their medical and surgical confrérès. This is because of the fact that in some hospitals the management

[1] Presidential Address delivered before the Twenty-fourth Annual Meeting of the Radiological Society of North America, at Pittsburgh, Dec. 1, 1938.

dominates the radiologist to the point where effective leadership cannot be maintained, and thus passes naturally to the other groups. Such men often lose their initiative for research and teaching and lapse into a state of apathy and become medical technicians only. These men cannot favorably influence ambitious young men to enter a specialty in which such conditions exist.

Next we must provide more uniformly adequate training for these men who select radiology for their lifework. There are at present many training centers which are doing an excellent job of turning out men with basic training and adequate experience to begin their lifework in this specialty. Unfortunately these training centers are too few.

Training centers should exercise great care in selecting young men of good character, and of proven industry and ability, in their interne work. They must not accept more men than their teaching material justifies, and, above all, these men must not be exploited as a source of cheap help under the guise of training. We do not mean to imply that they should not do considerable routine work but that most of it should be of a medical nature and under the direct supervision of the radiologist.

We believe that three years should be the minimum period of training and that during that time some provision should be made for instruction in pathology. The exact amount of time and effort spent on that subject will depend upon the facilities available. We feel that didactic instruction is of little value in this type of post-graduate teaching and that students must learn the technic of using the library in their search for information on specific subjects. Short discussions of the radiologic principles involved in cases seen during the daily work should stimulate the student to search extensively in the literature for other opinions on the same subject.

The students should be expected to attend the various hospital staff meetings and clinical–pathological conferences. They should also be present at joint conferences between the department of radiology and the various clinical departments, as such conferences are invaluable in correlating the roentgen findings with the history and clinical data.

The division of time between diagnosis and therapy must be worked out according to the division of the subjects in the department. In no instance shall the instruction in radiotherapy be sublimated to that of diagnosis. We believe that instruction in radium therapy should be carried out coincidentally with roentgen therapy, as the two supplement each other so closely in the treatment of disease processes.

Finally, we should encourage our students to engage in at least one piece of research work during the course of their training in order that they may have the advantage of the guidance and friendly criticism of their instructors. Radiologists must continue their research work in the future as in the past, and these young men must be the future leaders to carry the banners of progress.

The next subject which engages our interest is the improvement of the relation of the radiologists to hospitals. It is now acknowledged that the practice of radiology constitutes the practice of medicine and is not a part of the corporate activity of the hospital. This position must be zealously guarded so that we may not lose that identity, especially when group hospitalization attempts to use our services without remuneration. The radiologist must be an accredited department head on the same basis as other specialties and should not be attached to, or be a part of, some other department. This has proven to be a great handicap in the past in many departments.

The financial arrangement between the hospital and the radiologist has been a source of endless discussion. No one plan is applicable to all situations. It is obvious that the problem of the small hospital varies widely from that of the large one, and the large private hospital from the teaching hospital. In various instances, the arrangements are on a salary basis, commission basis, or rental basis, with satisfaction both to the radiologist and to the hospital. If each side is careful not to exploit the other, or the patient, any of these plans can be made to function satisfactorily. The Pacific Roentgen Club has for some time fought diligently for the rental plan, which has the additional advantage of preserving the private practice of radiology. The care of non-pay patients would seem to be one of the pitfalls of this plan and a very definite stipulation as to the number of these patients should be in the contract to avoid unjust accusations from either side. This is probably the most equitable plan that can be drawn up to safeguard the rights of both parties. In those institutions in which all mem-

bers of the medical staff are on salary, it is obvious that the radiologist should be compensated in like manner.

A pernicious attempt has been made to divide the radiologic service into so-called technical and professional portions. If this plan were carried out to its ultimate absurdity, almost every medical specialty would soon feel the iron heel of lay domination, and radiology would cease to be an independent medical specialty. Another problem which has caused radiologists grave concern has been the attempt to include radiologic services as insurance benefits in group hospitalization plans. Happily, the American Medical Association has recognized the threatened danger to the entire medical profession and has unequivocally denounced both plans. This has given us powerful support in the fight, but even at this time new attempts are constantly being made to include our services in insurance plans without our permission. This should and will be resisted with all the power of organized radiology.

We have discussed, thus far, the relation of the radiologist to the hospital, but the relation of the private practitioner of radiology to radiology in general is one of which we must not lose sight. The rights of these men, in relation to the general medical profession and to the hospital, must be carefully guarded if we are to preserve individualism in radiology. In the early days of radiology, individualism was the rule, and many of the men whose names shine brightest in radiologic history were always private practitioners of radiology. The work they did with the crude apparatus of their day is the envy of all of us. Let us strive to preserve the private practice of radiology so that individualism which has done so much to develop the science of our specialty shall not disappear from our ranks.

Finally, we must insist on our rights to be considered as consultants. In this way only can we render the ultimate in service to the patient and the physician. This is our strongest bulwark against the inroads of the commercial laboratory and the "picture taker." It is also our opportunity to educate the clinician in what radiology has to offer to the general medical profession, and to show that the trained radiologist is indispensable in the modern care of the sick. The radiologist, because of his wide interest in every medical specialty, is able to bring to the consultation a knowledge that often enables him to read values from the film which illuminate the situation with startling clarity.

Radiology is a living science and thus is constantly changing. We must be on the alert to see that it keeps pace with the scientific accomplishments of other medical specialties. A great deal has already been done within our own ranks to keep radiology abreast of modern medicine. The Board of Radiology is functioning in a manner that cannot fail to reflect credit on radiology, and this will be increasingly so in the coming years. The closer co-operation among the several scientific societies in radiology is bearing fruit and radiology was never so strong nor so respected as it is to-day. The Inter-Society Committee is attacking our economic difficulties with a vigor that promises to solve many of these vexatious problems for us. The educational programs which the various societies have initiated will be a great force in the future to elevate the standards of practice in radiology and I am happy that it has been possible for this Society to begin a series of instruction courses this year, which should lead to a new and better type of service which this Society can render to its members in the future. Serious thought should be devoted to the question of the desirability of devoting a greater percentage of time on our future programs to this type of instruction.

In closing, may I again urge that all the members of our Society put the weight of their experience and prestige behind the efforts which your Officers and Committees are making toward elevating the science of radiology and preventing inroads on our specialty by outside groups.

HOWARD P. DOUB, M.D.
Detroit, Michigan

PRELIMINARY PROGRAM

THE ANNUAL MEETING of the RADIOLOGICAL SOCIETY OF NORTH AMERICA

ATLANTA, GA., December 11–15, 1939

"A New Apparatus for Demonstrating Duodenal Ulcer." ARTHUR R. BLOOM, M.D., Detroit, Mich.

"Methods of Analyzing Cancer Statistics." LEWIS G. JACOBS, M.D., Winona, Minn.

"Solitary Myeloma of Bone: A Review of the Literature and Report of Four New Cases." L. W. PAUL, M.D., and E. A. POHLE, M.D., Ph.D., Madison, Wisc.

"Roentgenological Considerations in the Diagnosis and Treatment of Primary Malignant Bone Tumors." WILLIAM E. HOWES, M.D., and SAMUEL G. SCHENECK, M.D., Brooklyn, N. Y.

"Non-tuberculous Inflammatory Lesions in the Lungs." A symposium arranged by HOWARD P. DOUB, M.D., Detroit, Mich.

"Adult Hilum Tuberculous Adenopathy." B. P. WIDMANN, M.D., H. OSTRUM, M.D., and J. FETTER, M.D., Philadelphia, Pa.

"Aneurysm of the Splenic Artery." HARRY HAUSER, M.D., and J. V. SEIDS, M.D., Cleveland, Ohio.

"Ventricular Deformities Caused by Brain Tumors." ROBERT S. STONE, M.D., and NICHOLAS S. PEDERSEN, M.D., San Francisco, Calif.

"Intracranial Collections of Lipiodol Following Lumbar Myelography." L. H. GARLAND, M.D., San Francisco, Calif.

"Roentgen Diagnosis of Acute Abdominal Conditions." A symposium arranged by LEO G. RIGLER, M.D., Minneapolis, Minn.

"Nuclear Physics in the Service of Medicine." A symposium arranged by ROBERT R. NEWELL, M.D., San Francisco, Calif.

1. "The New Alchemy." M. A. TUVE, Washington, D. C.
2. "The Use of Radio-active Elements in Physiological Research." WILLIAM BALE, Rochester, N. Y.
3. "What does Artificial Radio-activity Promise for Therapy?" JOHN LAWRENCE, M.D., San Francisco, Calif.
4. "A Preliminary Report on the Skin Reactions of Patients Treated with Fast Neutrons Generated by the Cyclotron." ROBERT S. STONE, M.D., and JOHN LAWRENCE, M.D., San Francisco, Calif.

"Internal Hernia: Two Operatively Verified Cases of Paraduodenal Hernia." VINCENT W. ARCHER, M.D., and GEORGE COOPER, M.D., University, Va.

"Pendant Mastography." DAVID E. EHRLICH, M.D., New York City.

"Physician, Know Thyself." RALPH E. MYERS, M.D., Oklahoma City, Okla.

"LUCKENSCHADEL (caput fenistratum, lacunar skull)." E. C. VOGT, M.D., and GEORGE WYATT, M.D., Boston, Mass.

"Sarcoid, with Slides of Chest Findings over a Period of Two Years." E. R. BADER, M.D., Cincinnati, Ohio.

"Peptic Ulcer: A Review of 1,000 Cases and a Follow-up Study of the Patients Diagnosed between Ten and Twenty Years Ago." MAURICE F. DWYER, M.D., and WILLIAM S. COLE, M.D., Seattle, Wash.

"Cardiovascular Dynamics: A Roentgen Kymographic Study." SAMUEL BROWN, M.D., J. E. MCCARTHY, M.D., and ARCHIE FINE, M.D., Cincinnati, Ohio.

"Therapy Records as They are Kept and as They Should be Kept." ROBERT R. NEWELL, M.D., San Francisco, Calif.

"Myelography with the Use of Thorium Dioxide Sol as a Contrast Medium" (lantern demonstration). B. H. NICHOLS, M.D., and WILLIAM A. NOSIK, M.D., Cleveland, Ohio.

"Roentgen Treatment of Diphtheria Carrier." IRA I. Kaplan, M.D., New York City.

"The Use of 200,000 Volts in the Treatment of Advanced Superficial Malignancy" (illustrated with colored lantern slides). JOHN T. MURPHY, M.D., and C. E. HUFFORD, M.D., Toledo, Ohio.

"Diagnosis of, and Roentgen Therapy for, Myelogenous Leukemia." WALTER C. POPP, M.D., and CHARLES H. WATKINS, M.D., Rochester, Minn.

"Treatment of Different Types of Malignancies Occurring on the Skin of the Face, with Particular Reference to the Selection of the Type of Irradiation that will Give the Best Results." FRANKLIN B. BOGART, M.D., Chattanooga, Tenn.

"Rotation Therapy." SYDNEY J. HAWLEY, M.D., Danville, Pa.

"Pre-operative Irradiation of Breast Carcinoma." EDMUND P. HALLEY, M.D., and PERRY J. MELNICK, M.D., Decatur, Ill.

"Radium Treatment of Carcinoma of Corpus Uteri: Description of a New Applicator." MILTON FRIEDMAN, M.D., New York City.

"Primary and Secondary Filters in Roentgen Therapy." L. D. MARINELLI, New York City.

"Late Injuries Following Irradiation." ZOE A. JOHNSTON, M.D., Pittsburgh, Pa.

"Induction by X-rays of Hereditary Changes in Mice." GEORGE D. SNELL, Bar Harbor, Maine.

"Studies in Protection of Radium Therapy Personnel." ROBERT E. FRICKE, M.D., and MARVIN M. D. WILLIAMS, Ph.D., Rochester, Minn.

"Surface, Depth and Exit Doses for X-rays in the Range from 100 to 200 kv." EDITH H. QUIMBY, M.A., L. D. MARINELLI, and MANUEL GARCIA, New York City.

"X-ray Protection." LAURISTON S. TAYLOR, Ph.D., Washington, D. C.

"A Million-volt X-ray Unit." Dr. E. E. CHARLTON, Schenectady, N. Y.

"Accumulative X-ray Effects in Cells." P. S. HENSHAW, Ph.D., Washington, D. C.

"Osteochondrosis." C. A. STAMMEL, Lt. Col., M.C., U. S. Army, Fort Benning, Ga.

"Roentgen-ray Studies in Some Individuals Suffering from Low Back Pain: Lipiodol Studies before and after Operation." J. C. BELL, M.D., Louisville, Ky.

"Significance of Peristalsis of the Urinary Tract." J. P. KEITH, M.D., and E. L. SHIFLETT, M.D., Louisville, Ky.

"Sequelæ of Shoulder Dislocations." MAURICE D. SACHS, M.D., Portland, Oregon.

"Epithelioma of the Tonsil: Report of 162 Cases Treated with Radiation." W. L. MATTICK, M.D., Buffalo, N. Y.

"Contact Therapy." LOWELL S. GOIN, M.D., Los Angeles, Calif.

NEXT ANNUAL MEETING—REFRESHER SERIES

The schedule for the Refresher Series this year includes three hours Sunday afternoon, two hours Sunday evening, and two hours, from eight to ten, each morning of the Annual Meeting, December 11–15.

Several of the courses, particularly those subjects of wider scope, such as Radiology of the Gastro-intestinal Tract and Radiology of the Chest, are so arranged that related subjects will follow consecutively, making it possible to enroll in a sequential series. Basic presentations of Radiation Therapy will also be offered in series so that those with special interests may find Refresher Courses extending over the entire period of the series. The course on Physics of Radiation will be presented three hours on Sunday afternoon and two hours each morning of the meeting, and will follow the syllabus of lectures on the Physical Basis of Radiation Therapy, by Edith H. Quimby.

A formal announcement, with a description of each of the courses and its sequential relationship, will appear in the October issue of RADIOLOGY. There will be no enrollment fee. Members of the Society will be given preference and the number in the various courses will be limited by the size of the rooms available at the hotel.

Enrollment cards will be included in the annual letter which reaches the members of the Society thirty days prior to the Annual Meeting.

ATLANTA, PLACE OF 1939 ANNUAL MEETING[1]

It has been said that Atlanta owes its existence to railroads, but it is also true that other modes of transportation have aided the city in its amazing growth. When the state-owned railroad, in 1833, selected a terminal site in the foothills of the Blue Ridge Mountains, the growth of the community could not have been visualized or it would have been given another name than Terminus. From the one family that inhabited the wilderness when the railroad came, the population grew rapidly; in 1843 the name was changed to Marthasville and in 1847 to Atlanta. It became the State Capitol in 1869.

Many factors have contributed to the growth of the young city, which has a population of 402,450 people, and an historical setting that makes it a mecca for tourists. The population is almost entirely native-born, the foreign element constituting less than 2 per cent. When the motor age came, good roads were provided and now a well-equipped airport is in service for mail, passenger, and commercial use. The location of the city and the geographical formation of the country 'round about it, make it a natural gateway between the North and West to the entire group of Southeastern States. Therefore, one railroad followed another, until eight railroad systems, with fifteen mail lines, converged here.

Atlanta has an altitude of 1,050 feet, the highest city of its size, or larger, east of the Mississippi River. The mean annual temperature is 61 degrees, and there are no extremes of heat or cold. Few persons stop to think that Atlanta is only a little south of the halfway mark between New York and Miami. (It is 864 miles to New York and 715 miles to Miami.)

The city is a center of activities for the

[1] The factual statements in this story were furnished through the courtesy of the Atlanta Convention and Visitors' Bureau.

Aerial view of Atlanta.

Federal Government in the Southeast. It is the home of the Fourth Corps Area, Department of War, and 36 other branch divisions, not including the emergency governmental agencies. The Federal Penitentiary houses 2,000 offenders.

Atlanta is a city of beautiful homes and all the essentials that go to make living a joy and comfort. The city has parks and playgrounds covering approximately 2,000 acres, five municipally owned golf courses, and nine private clubs' courses. Tennis, boating, and swimming are sports provided at all the clubs and parks, and afford Atlanta visitors interesting pastimes.

The city is surrounded by colleges and universities, being the home of Oglethorpe University, Emory University, Georgia School of Technology, Agnes Scott College, Marist College, Woodbury Hall, Washington Seminary, and Georgia Military Academy. There are more institutions for higher learning for colored people in Atlanta than in any other city in the world. Six schools of college rank specialize in the education of the negro.

The visitors are always attracted by the points of interest in and around the city. Among these are: Wren's Nest, the home of Joel Chandler Harris, creator of "Uncle Remus"; the only exact replica of Bobby Burns' cottage in America; a cyclorama painting of the Battle of Atlanta, and Stone Mountain, a natural wonder, being five feet higher than the Woolworth Building.

The Radiological Society will hold its Annual Meeting at the Atlanta-Biltmore Hotel, from December 11 to 15, inclusive. Reservations should be made at once.

ANNOUNCEMENT

SIXTH INTERNATIONAL CONGRESS OF RADIOLOGY

Pursuant to the resolution passed in Chicago, in 1937, the Sixth International Congress of

The Atlanta-Biltmore Hotel, scene of the Next Annual Meeting of the Radiological Society of North America.

Radiology will take place in Berlin from July 31 to Aug. 4, 1940. It is proposed to use the Kroll Opera House for the sittings of the Congress. Members of radiological societies in all countries may become ordinary members of the Congress of Radiology, also persons recommended by radiological societies. As at previous Congresses, there will be general sessions and sectional sessions.

The following are some of the general subjects which are proposed for discussion: in diagnostics, "The modern development of special methods of examination in roentgen diagnostics and the results achieved with them" (automatism, cinematography, tomography, kymography, screen photography, contrast methods); in therapeutics, "The development of therapeutic methods, their achievements and the demarcation of their indications" (local and general radiation, quality of rays, time and space factors). It is further intended to give, in a series of connected lectures, a picture of the present state of knowledge as regards the effect of rays on the living cell (cell degeneration, mutations, impact theory), and also to make a report on the modern development of radiophysics. In addition, diagnostic and therapeutic results in individual spheres will be reported upon in connected lectures.

Beside the general lectures, departmental sessions will also be held, and the following subdivisions have been arranged for them: Roentgen diagnostics, radiotherapy, radiobiology, physics and technic, electrology and light.

Announcements of lectures must be sent to the President not later than Dec. 15, 1939. Extracts from papers, which should not exceed two typewritten pages of twenty-seven lines each, should be in the hands of the Secretary-

General not later than Feb. 1, 1940. The papers must be read by the author in person, who must be a member of the Congress.

Note concerning slides and films: Slides in the standard sizes $8^1/_2 \times 10$, 9×12, $3^1/_4 \times 4^1/_4$ can be shown. In the case of sizes other than these, it is necessary to send in an inquiry. Cinematographic films of 8 and 16 mm. widths can be projected.

The Rally is connected with an industry exhibition in the Zoo (inquiries should be sent to Mr. Kreykenbohm, Wirtschaftsgruppe Elektroindustrie, Fachabteilung "Elektromedizin," Berlin W 35, Corneliusstr. 3), and with a Book Show.

For all further details, you are requested to communicate with the Secretary-General. A detailed letter of invitation will be dispatched toward the end of the year.

Prof. Dr. H. Holthusen, *President*,
Hamburg 20, Goernestr. 29
Prof. Dr. W. Baensch, *Secretary-General*,
Leipzig C 1, Liebigstr. 20
Dr. med. h. c. B. Hauff, *Treasurer*,
Leipzig C 1, Rossplatz 12

COMMUNICATION

CANADIAN ASSOCIATION OF RADIOLOGISTS

The Canadian Association of Radiologists held its third Annual General Meeting at the Windsor Hotel, in Montreal, on June 22 and 23, with an attendance of between fifty and sixty members. The agenda of the Association was as usual limited to matters of business, the scientific sessions of Canadian radiologists being conducted through the Section of Radiology of the Canadian Medical Association. The main matters of discussion at the meeting were Relationship of Radiologists to Institutions in Contract Radiological Practice, and the matter of Inclusion of Medical Fees, Notably Radiological Fees, in Group Hospitalization Plan. Suitable resolutions were adopted by the Association and definite progress was made.

The membership of the Association now comprises 122, divided as follows: regular members, 98; members-elect, 16; associate members, 6; honorary members, 3. A. H. Pirie, M.D., of Montreal, was elected to Honorary Membership, the other two Canadian radiologists previously elected to Honorary Membership being M. H. Tovell, M.D., of Toronto, and Leo Pariseau, M.D., of Montreal. J. E. Gendreau, M.D., of Montreal, was elected the new President of the Association, W. H. McGuffin, M.D., of Calgary, Alberta, Vice-president, A. C. Singleton, M.D., of Toronto, was re-elected as Honorary Secretary-Treasurer. W. A. Jones, M.D., of Kingston, was elected Chairman of the Interrelations Committee.

IN MEMORIAM

WILLIAM J. MAYO, M.D.

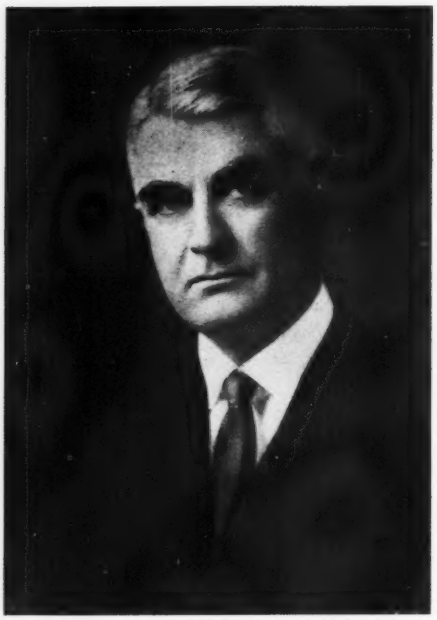
Dr. Mayo, master surgeon that he was, was so thoroughly in sympathy with the developments in radiology that he delivered the opening address before the Fifth International Congress of Radiology. He said in part:

"Medicine and surgery find themselves fortified by relatively new agents which day by day are being used increasingly in the treatment of disease. We recognize that in certain situations advanced cancer, which is beyond reasonable possibility of cure by the knife, can with but little risk be treated with radiant energy in the form of x-rays and radium, and even cured, if it has not progressed beyond the immediate site. We find that certain cellular tumors which are extremely active in growth can be treated with radiology with better results than with surgery, and without serious risk. This new agent, which began as an aid to surgery and medicine, extends its usefulness day by day."[1]

The biographical facts of Dr. Mayo's career are well known to all readers of RADIOLOGY, doubtless—in the main facts they parallel those in the life of his celebrated brother, Charles, who has just passed on. Let us, therefore, review briefly those incidents, honors, and interests which were peculiarly "Dr. Will's" own.

It has been said that William physically resembled the father, and he early adopted his father's way of life and some of his father's attitude toward life, which was expressed in the quotation, "He loved the truth and sought to know it." In his boyhood, too, he began to acquire those habits of industry that were to remain with him throughout life. At all events, he soon plunged into work; not odd jobs, such as most boys pick up, but medical

[1] RADIOLOGY, **30**, 647–649, May, 1938.



WILLIAM JAMES MAYO, M.D.
June 29, 1861–July 28, 1939

work of one kind or another. When he was still a schoolboy he studied chemistry and pharmacy and learned to compound prescriptions. . . . That attitude seemed always to have been his. As soon as he was old enough to do so he accompanied his father on his rounds. Before he entered high school he was assisting in the office, and occasionally at operations and postmortem examinations.

In the days when he was graduated from high school very little formal education was required preliminary to the study of medicine, and William was now eligible to enter medical school. His father, however, thought it wise for him to take further work in science and in languages. This the boy did, for three years, the first of which he spent in a private school especially devoted to the subjects named, and the last two of which he spent in Niles Academy. These years must have been most profitable, as much for the additional time it allowed for association with his father as for the curricular activities it allowed the youth to pursue. He was to see, in his freshman year in medical school, what an advantage his father's instruction in anatomy and microscopy had given him.

The formal medical course began, for W. J. Mayo, at the University of Michigan, in 1880. Some readers of this story may be unacquainted with the fact that the University of Michigan was founded as early as 1837, and that, by 1880, it had progressed so far that it was giving a three-year course in medicine instead of the customary two-year course. Great men were teaching in the medical school when W. J. Mayo matriculated there.

The three years passed rapidly and W. J. Mayo received his degree in 1883, when he was twenty-two years of age. He returned to Rochester to practise but, stimulated by his father, as well as following his own inclinations, he took every opportunity to go to other places to study, watch, listen, and make acquaintances. In 1884 he spent two months in the New York Post-graduate Medical School, and in 1885 he took a course in the New York Polyclinic, both of which awarded him, again, the degree of Doctor of Medicine. He also went to Philadelphia and to Chicago. When his brother came home, also ready to practise, they began a custom which they continued for years, namely, alternating in week-end trips to the surgical clinics of Chicago. As the years went on, the brothers included Europe in their purview, and they continued to travel about this hemisphere, and the other one, never satisfied that they, great as they were, knew it all. As before, they watched, listened, and made acquaintances, but in these latter days, no doubt, they contributed more than they took away.

Fifty-six years have passed since Dr. Will started to practise surgery. In that time he wrote nearly 600 papers. Dr. Will's enduring fame as a surgeon is assured. He was a master surgeon, and it is as a surgeon that the public thinks of him, for surgery is dramatic, and it is something that takes place behind closed doors; it has mystery.

In 1915 the brothers donated $1,500,000, with subsequent gifts now amounting to $2,800,000, to establish The Mayo Foundation for Medical Education and Research at Rochester, in affiliation with the University of Minnesota. In 1919 they founded The Mayo Properties Association, a charitable, scientific, and educational corporation, to hold all the properties, endowments, and funds of The Mayo Clinic, and to insure the permanency of the institution for public service. These

moneys and properties never can inure to the benefit of any individual. Records of deeds may become buried in archives, but the living institution which these men founded and the permanence of which they provided for, presumably will endure as long as Minnesota and the nation of which it is a part endure.

Scientific organizations made awards to Dr. Will. No detailed mention can be made here of the score or so of honorary academic degrees that were conferred on him. In addition to the posts of honor in foreign and American academic fraternities, Dr. Will was a member of twenty-seven medical organizations in this country and abroad.

Dr. Will was a great believer in the short siesta. He practised it himself for many years, and if that is the way to come through in as good condition as was his, for so many years, this country as a whole would do well to adopt the practice. Wherever he was, it was evident that Dr. Will's favorite companion was the calm, self-contained, dignified, great lady, Mrs. Mayo, who before her marriage, in 1884, was Miss Hattie Damon, of Rochester.

HENRY K. PANCOAST, M.D.

A fitting memorial to the late Henry K. Pancoast, M.D., is being prepared, to be published in the next issue of RADIOLOGY.

BOOKS RECEIVED

Books received are acknowledged under this heading, and such notice may be regarded as an acknowledgment of the courtesy of the sender. Reviews will be published in the interest of our readers and as space permits.

GRUNDLAGEN DER RÖNTGENDIAGNOSTIK UND RÖNTGENTHERAPIE (Principles of Roentgen Diagnosis and Roentgen Therapy). By DR. G. SCHULTE, Chief Physician of the Knappschafte Hospital in Recklinghausen and DR. F. KUHLMANN, of the medical clinic at the University of Halle. A monograph of 140 pages, with 148 illustrations. Published by Georg Thieme, Leipzig, 1939. Price: 8.50 R.M. bound.

CANCER OF THE COLON AND RECTUM: ITS DIAGNOSIS AND TREATMENT. By FRED W. RANKIN, B.A., M.A., M.D., Sc.D., F.A.C.S., Surgeon, St. Joseph's and Good Samaritan Hospitals, Lexington, Kentucky; and A. STEPHENS GRAHAM, M.D., M.S. (in Surgery), F.A.C.S., Surgeon, Stuart Circle Hospital, Richmond, Virginia, Assistant Professor of Surgery, Medical College of Virginia. A volume of 358 pages with 133 figures. Published by Charles C. Thomas, Springfield, Illinois, 1939. Price: $5.50.

ROENTGEN TECHNIC. By CLYDE MCNEILL, M.D., Louisville, Kentucky. A volume of 315 pages, with 268 illustrations and numerous tables. Published by Charles C. Thomas, Springfield, Illinois, 1939. Price: $5.00.

DIE HERZKRANKHEITEN, KLINIK, RÖNTGENBILD UND ELEKTROKARDIOGRAMM (Diseases of the Heart, Clinical, Roentgenologic, and Electrocardiographic aspects). By Dr. PAUL UHLENBRUCK, Professor in the University of Cologne; Chief Physician in the medical division of St. Elizabeth's Hospital and German Charity Institute for Health at Cologne. Second edition. A volume of 422 pages with 413 illustrations. Published by Johann Ambrosius Barth, Leipzig, 1939. Price: 38.00 R.M. bound (25 per cent discount allowed to foreign purchasers).

LEHRBUCH DER RÖNTGENOLOGISCHEN DIFFERENTIALDIAGNOSTIK DER ERKRANKUNGEN DER BRUSTORGANE (Roentgenologic Differential Diagnosis of Diseases of the Thoracic Organs). By Dr. WERNER TESCHENDORF, Chief Physician of the Radiation Institute, General Hospital, Cologne. A volume of 803 pages with 891 illustrations. Published by Georg Thieme, Leipzig, 1939. Price: paper, 69.00 R.M.; bound, 71.00 R.M. (25 per cent discount allowed to foreign purchasers).

RADIOLOGIE CLINIQUE DU COEUR ET DES GROS VAISSEAUX (Clinical Radiology of the Heart and Great Vessels). By C. LAUBRY, P. COTTENOT, D. ROUTIER, and R. HEIM DE BALSAC. Two volumes totalling 334 pages, with 1049 figures and drawings. Published by Masson et Cie, Paris, 1939. Price: Two volume export edition—430 francs.

L'ELECTRO ENCEPHALOGRAMME (Electro-encephalogram): NORMAL AND PATHOLOGIC. By IVAN BERTRAND, Director of the School of Hautes Etudes; JEAN DELAY, Hospital Physician, Hospital of Paris, and JACQUELINE GUILLAIN, Assistant in the Neurological Institute. A volume of 294 pages with 94 figures. Published by Masson et Cie, Paris, 1939. Price: 90 francs.

BOOK REVIEWS

LEUCHTSCHIRMPHOTOGRAPHIE RÖNTGENREIHENUNTERSUCHUNG (A Method of Roentgen Examination by Photographing the Shadow Structures on a Fluoroscopic Screen). By Prof. Dr. ROBERT JANKER, Bonn a. Rh. A volume of 57 pages, with 58 illustrations. Published by Johann Ambrosius Barth, Leipzig, 1939. Price: 9.00 R.M.

An excellent and rather comprehensive handling of the subject, especially as applied to pulmonary tuberculosis. Historical data are recorded since 1896 and emphasis is placed on work done since 1933. The author discusses the fundamental factors pertaining to fluorescent screen, lens, and micro-film adaptable to this work. He emphasizes its low cost for large group surveys, its economy in filing space and record keeping, and finally gives lay-outs for rooms and motorized trailers for portable work. His main development is toward the making of 24 × 24 mm. films of the chest, to be re-magnified for reading at a convenient size.

He does not anticipate that the micro-film will displace the standard full-sized film except for the case-finding programs of tuberculosis.

ERGEBNISSE DER GESAMTEN TUBERKULOSEFORSCHUNG (Results of the Entire Research on Tuberculosis). Edited by H. ASSMANN, H. BEITZKE, and H. BRAEUNING. A volume of 577 pages, with 135 illustrations and several colored plates. Published by Georg Thieme, Leipzig, 1939. Price: bound, 58.00 R.M. (25 per cent discount allowed to foreign purchasers).

This volume contains nine contributions prepared in monographic form and dealing with certain phases of tuberculosis. While undoubtedly the book is written primarily for the physician specializing in this field, several chapters contain sufficient references to diagnostic roentgenology and occasionally to radiation therapy to be of interest to the radiologist.

L. Hantschmann, who is working in the medical clinic of the University of Königsberg under the well-known H. Assmann, discusses a special type of sclerosing tuberculosis, which is histologically characterized by the presence of a peculiar large-celled hyperplasia described by Mylius-Schuermann and others. It produces a definite clinical picture as it involves the various organs, and in the lungs it simulates roentgenologically lymphangitis carcinomatosa. The essays on tuberculosis of the the nose and pharynx by F. Zöllner (Jena) and of the ear by A. Brüggemann (Giessen) both offer advice on the use of radiation in the treatment of these conditions. A chapter by E. Randerath (Düsseldorf) dealing with the pathological anatomy of tuberculosis of the larynx precedes the discussion of its clinical aspect by L. Rickmann (Ziegenhals). Of special interest are the relationship between tuberculosis of the larynx and lungs, the roentgen diagnosis of the laryngeal lesions, and the place of radiation in their treatment. Roentgen therapy in small doses (10 per cent S.E.D.) is beneficial and definitely analgesic. H. Schleussing (Munich) gives a good review of our present knowledge of the pathologic anatomy of tuberculosis of the urinary and male genital tracts, the clinical part being ably outlined by H. F. J. Weber (Vienna). While roentgen rays are now indispensable in the diagnosis of renal tuberculosis, they have not proved efficacious in its treatment. How valuable the proper use of roentgen rays as a guide in therapy can be is clearly shown in the chapter by H. Kleesattel (Rogzow) entitled "Extrapleural Pneumothorax and Oleo-pneumothorax." In the author's experience the method has proved especially advantageous in children. An analysis of the psychology of the tuberculous patient and steps to influence it, by H. Boening (Giessen) and H. Braeuning (Hohenkrug), points out the importance of adequate mental guidance in any program of anti-tuberculosis activities.

Each contribution is accompanied by a well-selected bibliography and the illustrations are excellent.

RÖNTGENATLAS DER ERKRANKUNGEN DES HERZENS UND DER GEFÄSSE. (Roentgen Atlas of Diseases of the Heart and Vessels.) Second edition. A monograph of 161 pages. Published by Urban & Schwarzenberg, Berlin, 1939. Price: 12.00 R.M. bound (25 per cent discount to foreign purchasers).

This interesting work is published primarily for the general practitioner. It is, as the title suggests, an atlas, containing 90 illustrations with an excellent text. The divisions of the book include an introduction, discussions of technics, the normal heart, the diseased heart, diseases of the pericardium, and diseases of the vessels. The subject matter is concerned with ordinary roentgenographic material, *i.e.*, the so-called six-foot heart radiograph, oblique views together with occasional lateral views and esophagograms. Kymography is referred to as more of an academic than as a practical procedure and is not stressed. The section on diseases of the blood vessels is confined to aortic diseases, and arteriosclerosis with calcification is seen as an incidental finding in other parts of the body. It is of interest that the

classic heart measurements (ML, MR, etc.) are mentioned only to minimize them since the author believes that there are many extraneous factors which alter their value. The variations of the normal heart are discussed quite extensively. The author is satisfied to refer to an increase of heart size from the general impression, rather than absolute measurement. Case histories, confirmed whenever possible, are used in presenting the material. Plastic models are used to illustrate morphologic changes in the cases discussed. The classic alterations in the heart shadow following valve changes incident to rheumatic heart disease are quite thoroughly shown. It is interesting to note that the author speaks rather glibly of a "mitral lung" which is a manifestation of increased lung markings due to greater pressure in the pulmonary circulation, occurring in mitral heart disease. Hypertensive and arteriosclerotic heart disease, syphilitic heart disease, and congenital heart disease are also discussed. The author feels that one cannot always make an absolute diagnosis in cases of congenital heart disease since there often is a multiplicity of lesions which produce bizarre changes in the cardiac silhouette. Cases of hyper- and hypothyroid heart disease and the rarer types of cardiac changes, such as seen in beri-beri, etc., are not discussed. The section on pericardial lesions is representative of the excellent manner in which the individual case histories are presented. As stated above, special technics are not introduced, hence arteriography is not mentioned, and the section on arterial disease thus is somewhat limited. One has the opinion after studying this worthwhile volume that there is a great deal of information which can be obtained by relatively simple radiographic methods which should be of value not only to roentgenologists but to cardiologists and general practitioners who are required to interpret their own roentgenographs.

ATLAS OF SKELETAL MATURATION. By T. WINGATE TODD, M.B., Ch.B. (Manc.); F.R.C.S. (Eng.). A volume of 202 pages, with 35 illustrations. Published by C. V. Mosby Company, St. Louis, 1937. Price: $7.50.

This work culminates one of the most painstaking and comprehensive studies of skeletal maturation as related to child development that has been undertaken. Doctor Todd and his associates in compiling this material reviewed thousands of roentgenograms and as is quite evident from the text, every possible care was used in determining and selecting the various age standards. The standards cover the period from three months after birth to the age of sixteen years in girls and nineteen years in boys, at which dates symbols or determinators of maturity provided by growing shaft surfaces, epiphyses related to these, and cartilage bones of the wrist cease to register progress.

The standards chosen to represent successive stages in maturity by intervals of three months to the end of the first year and by six-month intervals thereafter represent the accumulated roentgenographic records of Cleveland children from all grades of society, except the destitute, without regard to nationality of origin, type of family line, stature, and weight. The standards themselves are picked from groups of children of white stock ultimately of European origin. They are nevertheless applicable to children of negro parentage, for it has been shown that there is no practical distinction in determinators or in age relationships of maturation phase to differentiate white from colored children of similar social standing.

This volume should be of great interest to all those who are concerned with roentgenology of the skeletal system, for it will undoubtedly remain as an accepted standard for some time to come. The material is presented in a highly scientific manner and affords a great insight to a subject concerning which the average roentgenologist has a rather limited concept.

LEHRBUCH DER RÖNTGENDIAGNOSTIK (Textbook of Roentgenologic Diagnosis). By H. R. SCHINZ, W. BAENSCH, and E. FRIEDL. A text consisting of 2,182 pages, 2,810 illustrations, and 13 photographic plates, published as two volumes of two parts each, by Georg Thieme, Leipzig, 1939. Price: 270 R.M. bound (25 per cent discount allowed to foreign purchasers).

This the fourth edition of an internationally known comprehensive treatise of roentgenologic diagnosis that is rapidly taking on the cloak of a veritable encyclopedia concerning the subject. There is nothing comparable to it in any language at the present time. The present edition is entirely rewritten and reset and consists of 2,182 pages, an increase of

559 over the previous edition. The illustrations total 2,810, which is an increase of 560 over the last edition. In addition to Schinz, Baensch, and Friedl, the authors-in-chief, many chapters or special sections have been written by such well-known men as Franke, Holzmann, Hotz, Lindgren, Lysholm, Uehlinger, Ulrich, Weltz, and Zuppinger. It is impossible to single out any special section for praise or criticism for all are written in the most commendable manner. An outstanding feature of this edition is the great number of illustrations and drawings which are superbly reproduced. The distribution and character of many lesions of the skeletal and respiratory systems are shown by two- and three-color charts or drawings. There are thirteen full-page photographic plates.

It is unfortunate that the comprehensive bibliographies that were so useful in former editions have been omitted in this revision. Also notable are the few instances in which other authors in the literature are referred to in the text of a majority of the chapters. While the editors justify these omissions, the inclusion of a bibliography giving at least basic references would seem highly desirable in a reference work of this sort.

There have been frequent rumors in the past of an English translation of this work and, for the benefit of those who do not read German, such an edition would be very valuable. The text has become so large and the illustrations so numerous that the cost has become an appreciable figure; nevertheless, considering the scope of the work and the high quality of the illustrations and typography, it still remains a most desirable intellectual investment for those familiar with the German language.

ARQUIVO DE PATOLOGIA (Archives of Pathology). Orgao do Instituto Portugues Para o Estudo do Cancro. Edited by F. Gentil and M. Athias. Volume 11, No. 1, April, 1939. A volume of 200 pages, with numerous figures and tables in the text. Published by Oficina Grafica, Ltd., Lisbon, 1939. Price not stated.

This is a series of papers by experts in special lines of attack on cancer, each preceded by a résumé in Portuguese. Goyanes writes (in Spanish) about the Spanish league for the control of cancer, the activities of which were suppressed by the war. Simone Laborde writes two papers (in French), one on the results to be expected from the radiologic treatment of cancer, the other on carcinoma of the cervix. Cramer also contributes two papers (in French) on carcinoma of the stomach and on malignant tumors of the lung. Holthusen contributes one on the rôle of specialized institutions in combating cancer and one on the radiotherapy of laryngo-pharyngeal tumors (both in French). Then there are contributions by Lepierre on the Curies, da Silveira on waves and corpuscles, and by Guedes on Röntgen, the latter all in Portuguese.